EMERGENCY MEDICINE

Amit Agrawal
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Guide to Head & Spine Injuries

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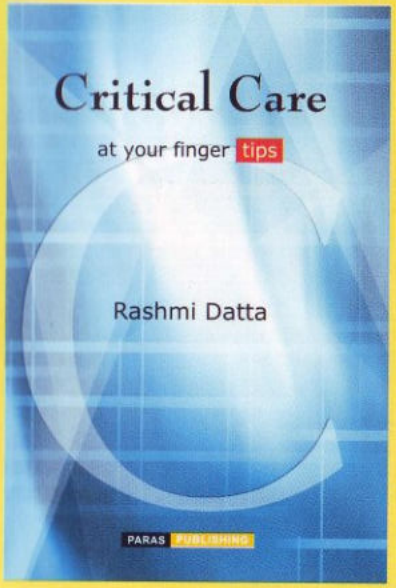

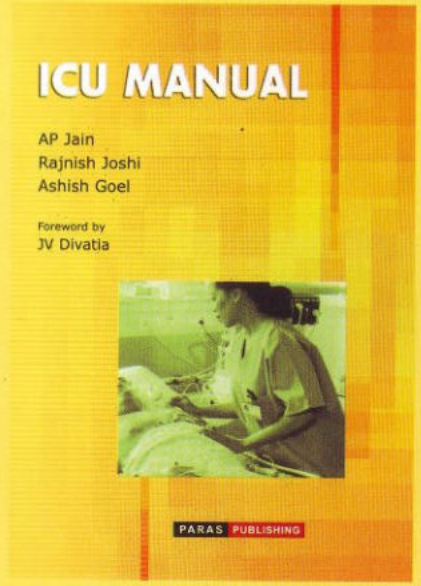

Guide to Head and Spine Injuries

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Paras Medical Publisher

Hyderabad, India

Guide to Head & Spine Injuries*

Published by:
Divyesh A Kothari
for **Paras Medical Publisher**
5-1-475, Putlibowli Road, PO Box 520
Hyderabad—500 095, India.
Tel: (040) 24742239
parasmedpub@hotmail.com

First Edition 2006

ISBN: 8 1-8191-131-8

Rs. 150:00

Printed in Hyderabad, India

Dedicated
to

My grandfather and grandmother

Foreword

It is indeed a pleasure to pen these words as a foreword to this book "*Guide to Head and Spine Injuries*" written by two very enthusiastic, innovative Neurosurgeons. Enthusiasm and innovation are what are required in the present arena of Medical Sciences with the fast pace of biotechnological breakthroughs we are facing every day.

We come across head and spine injuries every day in our practice and a basic understanding of these injuries is a must for every practising Surgeon and Physician. In these days where we depend so much on investigations and imaging techniques, this book I am sure will be a guide to the clinicians for proper assessment of patients to impart exact management.

Two of our younger colleagues have worked hard to simplify the assessment of these difficult injuries. I am sure the medical practitioners all over the world will utilize the depth of knowledge imparted in this book for better care of patients.

Let us march forward firm in our faith in learning, sustained in our confidence in the will of God and promise of young surgeons like Dr Amit Agrawal and Dr Dhaval P Shukla to make this world a happy place to live and let live.

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Preface

The social and technological advances in this country have put road traffic accidents on the forefront of health care delivery systems. As impact on head occurs in nearly 40% of all accidents, the management of head injuries is crucial in cases of road traffic accidents. Neurotrauma leads to special problems as the injuries to brain, spine and peripheral nerves can lead to severe disability. The training of neurosurgery is not universally available in all medical colleges, leading to several lacunae in management of neurotrauma at regional hospitals.

This book has been written in an attempt to educate persons involved in management of neurotrauma in places lacking advanced neurosurgical set up. The main purpose of this book is to make people aware how disabling neurotrauma can be and how early and protocol based management can save lives and reduce secondary complications so that when the patient reaches neurosurgeon he/she is in a state that treatment will lead to favourable outcome. The complicated details on exact mechanism of injuries, controversies in pathophysiology and management of injuries are not mentioned. Treatment protocols which can be instituted at primary trauma centre are emphasized. Chapters on prevention of injuries and rehabilitation are specifically added as they contribute significantly for management and can be easily instituted at centers with limited resources. After going through the book reader will be able to understand the injuries of head, spine and peripheral nerves in a better way and will be able to help the patients.

Amit Agrawal
Dhaval P Shukla

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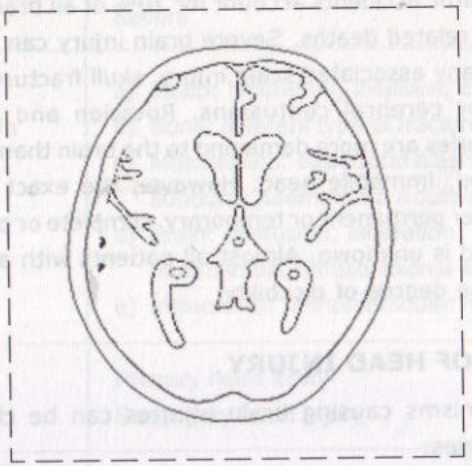


CHAPTER 1

Mechanism and Classification of Head Injuries

INTRODUCTION

Trauma is the leading cause of death in individuals under 45 years of age and road traffic accidents account for 70% of all brain injuries and 50% of trauma related deaths. Severe brain injury can occur even in the absence of any associated scalp injury, skull fracture, intracranial haemorrhage or cerebral contusions. Rotation and acceleration/deceleration injuries are more damaging to the brain than direct impact against the fixed, immobile head. However, the exact frequency of disability, whether permanent or temporary, complete or partial, among those discharged is unknown. Almost all patients with a severe head injury have some degree of disability.

MECHANISM OF HEAD INJURY

Physical mechanisms causing brain injuries can be classified into following categories:

Impact loading

Collision of the head with a solid object at a tangible speed. Impact loading causes brain injury through a combination of contact forces and inertial forces.

Impulsive loading

Sudden motion without significant physical contact.
Inertial force ensues when the head is set in motion with or without any contact force, leading to acceleration of the head.

Static or quasistatic loading

Loading in which the effect of speed of occurrence may not be significant. Contact force occurs when impact injury is delivered to the head at rest. Static or quasistatic loading is rare and occurs when a slowly moving object traps the head against a fixed rigid structure and gradually squeezes the skull, causing many comminuted fractures that may be enough to deform the brain. Contact or inertial forces may strain the brain tissue beyond its structural tolerance, leading to fatal injury. (Strain is the amount of tissue deformation caused by an applied mechanical force.)

Table 1.1 Classification of head injuries

Severity of injury	Mild Moderate Severe
Anatomical classification	a) Scalp: laceration, avulsion, contusion b) Bone: different type of fractures c) Membranes: extradural haematoma, subdural haematoma acute/chronic d) Brain: contusion, laceration, intracerebral haematoma, diffuse axonal injury e) Intracranial: intraventricular haemorrhage
Duration since impact	Primary head injury Secondary head injury
Extent of involvement	Focal Diffuse
Pathological classification	Concussion Contusion Intracranial haematomas Traumatic subarachnoid haemorrhage Skull fractures Diffuse axonal injury
Site of impact	Coup, Contrecoup

Three basic types of tissue deformation include the following:

- Compressive: Tissue compression
- Tensile: Tissue stretching
- Shear: Tissue distortion produced when tissue slides over other tissue

Classification (Table 1.1)

Head injuries can be classified according to severity of the injury, anatomical structures involved, depending on duration since impact (primary and secondary), extent of involvement, underlying pathology or site of impact (Table 1.1). Following terms need futher elaboration for better understading:

Primary Head Injury (Table 1.2)

Primary head injury is the mechanical trauma that occurs at the moment of impact and may lead to irreversible cell damage from physical disruption of neurons or axons. The primary injury is well established and irreversible by the time the patient gets to the hospital (Table 1.2).

Secondary Head Injury (Table 1.3)

Secondary head injury is any physiological event that can occur within minutes, hours, or days after the initial injury and leads to further damage of nervous tissue. Such secondary insults may contribute to permanent neurological dysfunction. Meaningful recovery of function after head injury is possible if these secondary insults are prevented or minimized. Preventing secondary injury is the most important aspect of trauma care. For this, prompt diagnosis & treatment are vital

Table 1.2 Characteristics of primary head injury

- Damage caused at time of impact
- Can be focal or diffuse
- Diffuse axonal injury is due to deceleration and shearing forces
- Depends on extent of initial injury
- Occurs almost immediately at the trauma site
- Direct insult to the brain and skull leading to decreased tissue perfusion, ischaemia and infarction
- Difficult to treat

Table 1.3 Systemic and intracranial complications that result in additional damage to the brain

Systemic causes	Intracranial causes
Hypoxaemia	Increased ICP
Anaemia	Cerebral oedema
Hypotension	Mass lesions
Hyperthermia	Infection
Hypercapnoea	Seizures
Hypocapnoea	
Respiratory complications	
Electrolyte imbalances	

Focal Injury

Focal injury can occur anywhere but it is more commonly seen in frontal and temporal lobes because of the shape of inner surface of the skull. This is the result of deformation of brain at the point of impact, by a small object such as a hammer, or a rock. This results in injury to the scalp, skull fracture and could be associated with dural laceration and underlying brain contusion or laceration. Usually there is a localized surrounding oedema around the site of the impact.

Diffuse Injury

Diffuse injury (referred to as diffuse axonal injury or DAI) is only visible on CT scan in the worst 5% of cases, and most commonly seen as multiple punctate subcortical lesions in and around the corpus callosum and deep white matter and/or as intraventricular haemorrhages. The most consistent effect of diffuse brain damage, even when mild, is the presence of altered consciousness. The depth and duration of coma provide the best guide to the severity of the diffuse damage. The majority of patients with DAI will not have any CT evidence to support the diagnosis. Other clinical markers of DAI include high speed of injury, absence of a lucid interval, and prolonged retrograde and anterograde amnesia.

Coup and Contrecoup Injuries

The amount of energy dissipated at the site of direct impact determines whether the ensuing contusion is of the coup or contrecoup type. CSF plays a major role in coup and countercoup injuries to the brain. A blow to a stationary but moveable head causes acceleration, and the brain floating in CSF lags behind, sustaining an injury directly underneath the point of impact (coup injury, Figure 1.1). Most of the energy of impact from a small hard object tends to dissipate at the impact site, leading to a coup contusion. Scalp injuries (bruise, abrasion or laceration), contusion or laceration of the brain surface at the site of a fracture, especially if it is depressed are examples of coup injuries. When a moving head hits the floor, sudden deceleration results in an injury to the brain on the opposite side (contrecoup injury, Figure 1.2).

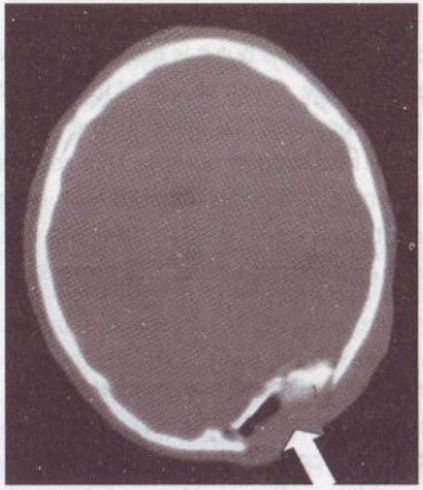


Fig. 1.1 Coup injury. Depressed fracture at the site of impact (arrow)

In contrast to coup injuries, impact from a larger object causes less injury at the impact site since energy is dissipated at the beginning or end of the head motion, leading to a contrecoup contusion. Such contrecoup contusions occur where the brain glides over the irregular, jagged contours of the skull interior and are usually more severe than the corresponding coup-type contusions. Negative suction pressure which develops opposite to the point of impact is also involved in their causation. Greater contrecoup forces may also lacerate the brain surface. In this way a backwards fall causes contrecoup contusions at the front of the brain (to the frontal and temporal poles).

Similarly, a fall onto one side of the head causes contrecoup contusions at the opposite side of the brain (temporal lobe). At autopsy, this pattern is useful in distinguishing head injuries due to falls from those due to blows. However, a forward fall does not cause contrecoup contusions over the back of the brain due to the interior of the skull being smooth at this point.

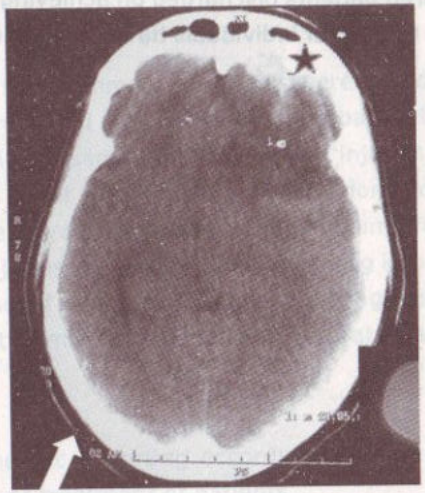


Fig. 1.2 Contrecoup injury. Contusion (star) diagonally opposite to the site of impact (arrow)

FACTS AND FIGURES

- Brain injury is 10 times more common than spinal injury.
- Majority of deaths in severe head injury occur before reaching to hospital.
- Head injuries are twice more common among males compared with females.
- Pedestrian injuries account for about 15% of brain injuries.
- Motorcycle-related deaths occur at a rate that is 15 times greater than the rate for occupants of passenger cars.
- Over half of all motorcycle crash-related deaths involve a head injury.

- Laws that require helmet use have been shown to reduce deaths in motorcyclists by about 30%.
- Alcohol intoxication is a compounding factor in at least 30% to 50% of head injuries and is a contributing factor in almost one half of all fatal motor vehicle crashes.
- Age and severity of brain injury have been shown to be consistent and major predictors of recovery from brain injury.
- The amount of disability among those with moderate brain injury is about 2 in every 3 persons.
- For head injured patients discharged on achieving a good recovery, at least 10% to 20% of individuals have ongoing neuropsychological problems.

CHAPTER 2

Evaluation of Head Injuries

Head injuries are very common and when severe they can be distressing to manage. A structured and systematic approach will avoid errors in these patients. At the same time less severe injuries will also require skill if pathology is not to be missed. Finally, don't forget that record keeping must be beyond reproach as legal claims for compensation following head injuries are common. Before going into details of clinical evaluation we should know few things about triage as this is helpful in the management of mass casualties and natural disasters.

TRIAGE

Triage means the act of sorting according to degrees of urgency to decide the order of treatment of patients.

The Objectives of Triage

To prioritize patients with a high likelihood of early clinical deterioration. To maximize the number of patients who are salvaged under the prevailing conditions of limited resources. However, it can result in bypassing seriously injured patients while less critical patients have been stabilized.

Protocol for Triage (Table 2.1)

Protocol for initial evaluation is designed to identify life-threatening injuries and to initiate stabilizing treatment in a rapidly efficient manner. Absolute diagnostic certainty is not required to treat critical clinical conditions identified early in the process. When resources are limited (e.g., one physician), do not perform subsequent steps in the primary

survey until after addressing life-threatening conditions in the earlier steps.

Table 2.1 Protocol for triage

- Initial evaluation
- Primary survey
- Resuscitation
- Secondary survey
- Definitive treatment or transfer to an appropriate trauma centre for definitive care

Triage Categories (Table 2.2)

It is performed by a triage officer who assesses casualties without giving treatment and divides patients into categories. Casualties may be given coloured triage label and managed accordingly.

Table 2.2 Description of triage categories

Category	Definition	Colour	Treatment	Example
P1	Life-threatening	Red	Immediate	Tension pneumothorax
P2	Urgent	Yellow	Urgent	Fractured femur
P3	Minor	Green	Delayed	Sprained ankle
P4	Dead	White		

Method of Triage

Triage can be performed rapidly by assessing:

- Ability to walk
- Airway
- Respiratory rate
- Pulse rate or capillary return

Rapid Evaluation

Patients details can be noted rapidly by considering the following points and SIMPLE history (Box) can be noted:

- Vital signs
- Prehospital clinical course
- Mechanism of injury
- Patient age
- Known or suspected comorbid conditions

The simple history

- S—Symptoms
- I—Injury details
- M—Patient medication history
- P—Past medical and surgical history
- L—Time of last meal
- E—Full description of events

Important Considerations

Patients with following findings will need an accelerated workup for better outcome:

- Multiple injuries
- Extremes of age
- Evidence of severe neurologic injury
- Unstable vital signs
- Preexisting cardiac or pulmonary disease

Remember

When performing a triage with patients having different types of injuries, the priorities of the primary survey help to determine precedence (e.g., a patient with an obstructed airway receives greater priority for initial attention than a relatively stable patient with a traumatic amputation). In trauma centers, a team of providers evaluates patients who are critically injured and simultaneously performs diagnostic procedures. This parallel processing approach can dramatically reduce the time required to assess and stabilize a patient with multiple injuries. The team approach to trauma is resource intensive; however, the available personnel and resources can become overwhelmed quickly in nonhospital settings, in smaller institutions, and in mass casualty situations. Under these conditions, additional factors affect the triage process, including the number and skill levels of available providers, the available equipment, and the provider estimate of the clinical probability of each patient survival. Regardless of the clinical setting,

organize the care team prior to patient arrival. Leadership and unity of command are essential for directing a rapid and efficient workup. In larger institutions with dedicated trauma services, general surgeons form the core of the trauma team in close cooperation with the emergency department staff. A physician from either service who is experienced in the care of trauma patients serves as the team leader and directs evaluation and resuscitation. Additional physicians or mid-level providers are responsible for managing the airway, conducting the primary and secondary surveys, and performing other procedures as needed. Nurses and technicians monitor vital signs, gain intravenous (IV) access, and obtain blood samples. Respiratory therapists and radiology technologists also should be present. As consultants, neurosurgeons and orthopaedic surgeons must be available immediately to the trauma team. Early consultation with a neurosurgeon is mandatory when significant central nervous system injury is present. Specific procedures performed by both neurosurgeons and orthopaedician can be life saving.

CLINICAL EVALUATION

PATIENT HISTORY

A thorough neurological history allows the clinician to define the patient problem and, along with the physical examination, assists in formulating an aetiologic/pathologic diagnosis in most cases. Neurological examination begins with the gathering of an accurate patient history and information about the course of the present injury. This will help to create a baseline as well as to provide valuable information about the course and characteristics of the present illness. In emergency situation the patient history should include a brief personal profile and description of the patient. A repeated systemic approach will avoid missing the details and errors. A simple and comprehensive scheme as outlined below can be followed and modified according to the emergency situation.

History of the Presenting Illness and Chief Complaints (Table 2.3)

History of present illness includes onset of the problem, the setting it developed in, manifestations and past treatment for the problem and in addition it includes details of precipitating event

- Symptom onset (acute, subacute, chronic, insidious)
- Duration of symptoms
- Course of the condition (static, progressive, or exacerbating and remitting).
- Associated symptoms such as seizures, headache, nausea, vomiting, and pain

Table 2.3 Salient features to consider in the history of patient with head injury

- Type of accident
- Mechanism of injury
- High or low velocity injury
- Loss or alteration of consciousness
- Post-traumatic amnesia and retrograde amnesia
- Vomiting
- Epileptic fits or seizures
- Swelling and pain in head
- Other complaints: visual, hearing or balance disturbances

Past History

- Past accidents and injuries
- Operations
- Previous hospitalizations

Personal History

- Current health
- Allergies
- Any history of alcohol intake or smoking

Family History

A brief family history should be included, ***the source of the information indicated*** and the mental status of the patient to be noted.

Medication Review

Ask the details of over-the-counter and prescription medications as well as use of recreational drugs.

GENERAL EXAMINATION OF THE HEAD INJURIES

General examination will follow the guidelines as for any other discipiline of medicine with more emphasis on head, spine and neurological details.

Pulse and Blood Pressure

Note for any bradycardia or tachycardia. Bradycardia with hypertension is a late sign of raised intracranial pressure (Cushing's triad—bradycardia, hypertension and raised ICP). Tachycardia with hypotension in head injury patients is a sign of blood loss. In these cases look carefully for any obvious or occult (chest, long, bone or abdomen) blood loss. Check for hypothermia or hyperthermia and avoid exteme changes in body temperature.

Respiration

Abnormalities of the rate, rhythm, and amplitude of respiration may occur in patients of head injury due to central nervous system dysfunction.

Biot's respiration (ataxic breathing)—Abrupt and irregularly alternating periods of apnoea with periods of breathing that are consistent in rate and depth, often the result of increased intracranial pressure.

Cheyne-Stokes respirations—A breathing pattern characterized by a period of apnoea, followed by gradually increasing depth and frequency of respiration.

Kussmaul breathing—A very deep gasping type of respiration associated with severe diabetic acidosis and coma.

Position of the Patient

Look for any abnormal posturing (decerebrate or decortical rigidity).

Head and Neck

- Inspect the whole head and look for scalp bleeding (rapidly suture briskly bleeding lesions).
- Palpate for facial fractures and a mobile middle third of the face.
- Basal skull fractures are suggested by a haemotympanum and the presence of bruising around the eyes (i.e., raccoon eyes) and ears (i.e., Battle sign).
- Look for any bleeding from the nose, ear or mouth.
- Presence of neurological deficits.
- Neck: presence of any tenderness or bruises.
- Carotids: Auscultate carotids for any bruit.

Other CNS Manifestations

- Look for any CSF otorrhoea or rhinohrrhoea

Review of Other Major Systems

Ask the patient about any problems with the other major systems of the body including heart, lungs and abdomen. Exclude any fracture ribs, haemopneumothorax, cardiac injury or haemoperitoneum.

Tetanus prophylaxis—Do not forget to give tetanus prophylaxis.

NEUROLOGICAL EXAMINATION

A sytematic and careful neurological examination is mandatory for the management of patients with head injury. To perform this, one also needs some basic instruments (Table 2.4). All clinical details and findings should be carefully noted and analyzed at the end of examination to make a diagnosis and to plan out a management protocol for the patient.

Table 2.4 Tools necessary for neurological examination

- Reflex h ammer
- Penlight
- Tongue b lade
- Safety pin
- Cotton s wab
- Ophthalmoscope
- A Snellen eye chart or pocket vision card
- 128 and 512 (or 1024) Hz tuning forks
- Dermatome c hart

Level of Consciousness

Level of consciousness can rapidly be assessed by simple method devised by Australasian College of Surgeons (Table 2.5); however, Glasgow coma scale is the most acceptable and reproducible method to assess the level of consciousness. It is easy to use and can be performed and reproduced with simple skills. The Glasgow Coma scale (Table 2.6) was first developed in 1974 as a way to assess and monitor levels of consciousness.

Table 2.5 Simple method to assess level of consciousness (Australasian College of Surgeons)

Alert	Normal response
Obtunded or confused	A slight but noticeable decrease in alertness with decreased interest in what is happening in the environment, decreased attention span & memory
Stupor	The person appears to be in deep sleep but can be aroused by noxious or vigorous stimuli
Coma	Eyes closed, no directed motor or verbal activity and unarousable

Table 2.6 Glasgow coma scale

Parameter	Response	Score
Eye opening	4 3 2 1	Spontaneous To voice To pain No response
Best verbal response	5 4 3 2 1	Oriented, converses Disoriented, converses Inappropriate words Incomprehensible sounds No response
Best motor response	6 5 4 3 2 1	Follows commands Localizes Withdraws Abnormal flexion Abnormal extension No response

Remember that minimum score is 3 and maximum possible score is 15 in this system and best possible score is noted in response to stimuli. Painful stimuli should be given in the distribution of cranial nerves (i.e., pressure on the supratrochlear nerve and ear lobule) to avoid spinal reflexes. A Glasgow score of 8 or less out of a possible score of 15 defines coma in 90% of cases.

Pupillary Examination

A careful pupillary examination is a critical part of the evaluation of patients with head injury, especially in patients with severe injuries. When muscle relaxants have been administered to a patient, the only aspect of the neurologic examination that may be evaluated is the pupillary examination.

Method to test pupillary reactions to light

1. Dim the room lights as necessary.
2. Ask the patient to look into the distance (if patient is co-operative).
3. Shine a bright light obliquely into each pupil in turn.
4. Look for both the direct (same eye) and consensual (other eye) reactions.
5. Record pupil size (in mm), any asymmetry or irregularity.

Interpretations of pupillary reaction

Proper assessment of the pupillary response requires the use of a strong light source to override any of the potential factors that may affect pupillary reaction. Each pupil must be assessed individually, with at least 10 seconds between assessment of each eye to allow consensual responses to fade prior to stimulating the opposite eye.

Normal pupillary examination. A normal pupillary examination result consists of bilaterally reactive pupils that react to both direct and consensual stimuli.

Bilateral constricted pupils. Narcotics cause pupillary constriction (miosis), and medications or drugs that have sympathomimetic properties cause pupillary dilation (mydriasis). These effects are often strong enough to blunt or practically eliminate pupillary responses.

Prior eye surgery, such as cataract surgery, can also alter or eliminate pupillary reactivity. Bilateral small pupils can be caused by narcotics, pontine injury (due to disruption of sympathetic centers in the pons), or early central herniation (mass effect on the pons).

Bilateral dilated and fixed pupils. Bilateral dilated and fixed pupils are secondary to inadequate cerebral perfusion. This can result from diffuse cerebral hypoxia or severe elevations of ICP preventing adequate blood flow into the brain. Pupils that are fixed and dilated usually indicate an irreversible brain injury. If it is due to systemic hypoxia, the pupils sometimes recover reactivity when adequate oxygenation is restored.

Unilateral dilated and fixed pupil. A unilateral dilated and fixed (unresponsive) pupil has many potential causes. A unilateral dilated pupil that does not respond to either direct or consensual stimulation usually indicates transtentorial herniation. A pupil that does not constrict when light is directed at the pupil but constricts when light is directed into the contralateral pupil (intact consensual response) is indicative of a traumatic optic nerve injury.

Unilateral constricted pupil. Unilateral constriction of a pupil is usually secondary to Horner syndrome, in which the sympathetic input to the eye is disrupted and the pupil constricts due to more parasympathetic than sympathetic stimulation. In patients with head injury, Horner syndrome may be caused by an injury to the sympathetic chain at the apex of the lung or a carotid artery injury. A unilateral constricted pupil can be caused by a unilateral brainstem injury, but this is quite rare.

Core optic pupil. A core optic pupil is a pupil that appears irregular in shape. This is caused by a lack of coordination of contraction of the muscle fibers of the iris and is associated with midbrain injuries.

Ocular Movements

When the patient level of consciousness is altered significantly, a loss of voluntary eye movements and presence of abnormalities in ocular movements are suggestive of brain stem dysfunction. These abnormalities can provide specific clues to the extent and location of injury.

Ocular movements involve the coordination of multiple centers in the brain, including the frontal eye fields, paramedian pontine reticular formation (PPRF), medial longitudinal fasciculus (MLF), and nuclei of the third and sixth cranial nerves. In patients in whom voluntary eye movements cannot be assessed, oculocephalic and oculovestibular testing may be performed.

Check for the Position of the Eyes in Primary Gaze

By having the patients look straight ahead, shine a bright light into the pupils. Reflection of light should be same in each pupil. A deviation suggests presence of a strabismus (inability to align the visual axes so they are directed at the same point). That may be due to weakness of extraocular muscle (i.e., weakness of lateral rectus muscle will cause deviation of eye medially).

Oculocephalic Testing

Before performing oculocephalic testing, status of the cervical spine must be established. If a cervical spine injury has not been excluded reliably, oculocephalic testing should not be performed.

Oculocephalic testing (doll's eyes) involves observation of eye movements when head is turned from side to side. This maneuver helps assess the integrity of horizontal gaze centers. To assess oculocephalic movements, head is elevated to 30° from horizontal and is rotated briskly from side to side.

Interpretation

Normal response. A normal response for eyes is to turn away from direction of movement as if they are fixating on a target that is straight ahead. This is similar to the way a doll's eyes move when head is turned; this is origin of the term doll's eyes.

Abnormal response. If eyes remain fixed in position and do not rotate with head, is indicative of dysfunction in lateral gaze centers and is referred to as negative doll eyes.

Oculovestibular Testing

Oculovestibular testing requires the presence of an intact tympanic membrane; this must be assessed before performing the test.

Oculovestibular testing, also known as cold calorics, is another method for assessment of integrity of the gaze centers (brainstem function). Oculovestibular testing is performed with the head elevated to 30° from horizontal to bring horizontal semicircular canal into vertical position. In oculovestibular testing, 20 mL of ice-cold water is instilled slowly into the auditory canal. If is no response occurs within 60 seconds, test is repeated with 40 mL of cold water. When cold water is irrigated into the external auditory canal, temperature of endolymph falls and fluid begins to settle. This causes an imbalance in vestibular signals and initiates a compensatory response. Cold-water irrigation in ear of an alert patient results in a fast nystagmus away from irrigated ear and a slow compensatory nystagmus toward the irrigated side. If warm water is used, opposite will occur; fast component of nystagmus will be toward the irrigated side, and the slow component will be away from the irrigated side. This is the basis for the acronym COWS, which stands for cold opposite, warm same. This refers to the direction of fast component of nystagmus. As the level of consciousness declines, fast component of nystagmus fades gradually. Thus, in unconscious patients, only slow phase of nystagmus may be evaluated. Some patients may have negative doll's eyes and normal oculovestibular reflexes.

Interpretation

Normal oculocephalic response. A normal oculocephalic response to cold-water calorics (i.e., eye deviation toward the side of irrigation) indicates that injury spares PPRF, MLF, and third and sixth cranial nerve nuclei. This means that the level of injury must be rostral to the reticular activating system in upper brainstem.

Unilateral frontal lobe injury. If a unilateral frontal lobe injury is present, the eyes are deviated toward the side of injury prior to caloric testing. Cold-water irrigation of the opposite ear results in a normal response to caloric testing (i.e., eye deviation toward the irrigated side) because the injury is in the frontal region and spares the pontine gaze centers.

Pontine injury. When a pontine injury is present, eyes often deviate away from the side of injury. In this situation, cold-water irrigation of contralateral ear does not cause gaze to deviate towards irrigated ear because an injury has occurred at the level of pons and pontine gaze centers are compromised.

Dysconjugate response. A dysconjugate response to caloric testing suggests an injury to either third or sixth cranial nerves or an injury to MLF, resulting in an internuclear ophthalmoplegia.

Skew deviation. If caloric testing causes a skew deviation, in which eyes are dysconjugate in vertical direction, indicates a lesion in the brainstem. The exact location of injury that results in skew deviation is not known.

Motor Examination

After completing brainstem examination, a motor examination should be performed. However, a thorough motor or sensory examination is difficult to perform in any patient with an altered level of consciousness. When a patient is not alert enough to cooperate with strength testing, motor examination is limited to an assessment of asymmetry in findings. This may be demonstrated by an asymmetric response to central pain stimulation or a difference in muscle tone between left and right sides. A finding of significant asymmetry during motor examination may be indicative of a hemispheric injury and raises possibility of a mass lesion. Completion of a neurological evaluation on a person with a suspected neurological injury will give a picture of the extent and possible location of nervous system damage. This is especially important with sudden onset or life-threatening diseases in which the outcome is dependent on an accurate referral or timely treatment. With practice, one will be able to refine his evaluation skills and will be able to complete the neurological exam quickly and efficiently. Details of motor, sensory and reflex examination are further discussed in the section of spine injuries.

INVESTIGATIONS IN HEAD INJURIES

Lab Studies

Haemogram

A complete blood cell count, including platelets, provides a baseline haematocrit and should be monitored serially, especially when bleeding is suspected.

Blood chemistry

Blood chemistry, including an amylase and lipase, provides information regarding other organ injury. Random blood glucose should be determined and maintained at normal level. Both hypoglycaemia and hyperglycaemia are harmful to brain.

Coagulation profile

Coagulation profile, prothrombin time (PT)/activated partial thromboplastin time (aPTT), and fibrinogen should be obtained in patients with head trauma because they may have an underlying or trauma-triggered coagulopathy particulary in patients with cerebral contusions and diffuse brain injuries.

Blood grouping and cross matching

Type and cross matching is useful in anticipation of need for transfusion, especially in patients with multiple traumas.

ABG

Arterial blood gas provides information regarding oxygenation and ventilation, and results will help in correction of any ventilatory insufficiency.

Toxicology

A blood or urine toxicology screen should be obtained in addition to the routine panel, especially in patients who have altered mental status, seizures, and an unclear history.

Wound swab

Wound cultures from lacerations or open skull fractures should be taken; findings willl help to choose appropriate antibiotics when infection is suspected.

Plain Skull x-ray

Skull x-rays are not much of help in the management of head injuries. x-rays are indicated in conscious but symptomatic patient with head injury when no other facility is available and minimum two views—AP and lateral—should be taken (Table 2.7).

Table 2.7 Required views

- Lateral (right or left according to site of injury)
- Antero-posterior
- Half axial (Towne's) view
- PNS view

Differential diagnosis of intracranial calcification and skull radiolucencies are shown in Table 2.8 and Table 2.9 respectively. If X-ray shows fracture then patient should be further investigated with CT scan.

Table 2.8 Causes of intracranial calcification

Normal	Pineal g land Dual (commonly falx) Vascular (carotid arteries) Basal g anglia
Abnormal	Tumours: c raniopharyngioma, m eningioma Arteriovenous m alformations Aneurysms Vault or sinus osteomas

CT scan

CT is a vital tool in assessment of patients with serious head injuries or patients with unstable multiple organ injuries, and it has revolutionized the management of these patients (Table 2.10). It remains the investigation of choice even following the advent of MRI, due to the ease of monitoring of injured patients and better demonstration of intracranial pathology and bony injury (Table 2.11).

Table 2.9 Differential diagnosis of skull radiolucencies

Normal	Squamous temporal bone
	Pacchionian granulations
Air	
Superficial	After scalp injury
Intracranial	After open fracture
Outer skull table	Rodent ulcer
Inner skull table	Slow growing tumours
	Chronic subdural haematoma
Diffuse lesions	Metastases
	Paget's disease
	Multiple myeloma
	Hyperparathyroidism

A non-contrast study is useful in immediate post-trauma period for rapid diagnosis of intracranial pathology that may require prompt surgical intervention. To review CT scan, a systematic approach, consistency and specific order with same protocol each time are important (Figure 2.1).

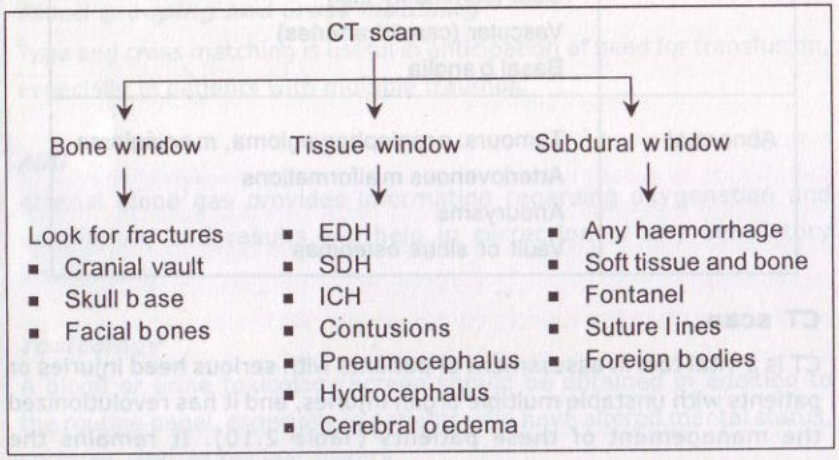


Fig. 2.1 Systematic approach to read CT scan

MRI

MRI has a limited role in the evaluation of acute head injury but it is not practical in emergency situations because magnetic field precludes the use of monitors and life-support equipments needed by unstable patients. MRI is a more sensitive imaging study providing more detailed information regarding the anatomic & vascular structures, and detection of small haemorrhages in areas that might escape CT scanning.

Table 2.10 Indications for computed tomography scanning

- GCS score < 15 at 2 hours after injury
- Vomiting > 2 episodes
- Age > 65 years
- Amnesia before impact > 30 minutes
- Dangerous mechanism (pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from height > 3 feet or 5 stairs)
- Confusion or worse, impairment of consciousness, epileptic seizure, neurological symptoms or signs
- Deteriorating consciousness or progressive neurological deficits
- Penetrating injury
- Posttraumatic seizures
- Confusion (GCS < 14) persisting after initial assessment and resuscitation
- Progressive headache
- Unreliable history or examination because of possible alcohol or drug ingestion
- Loss of consciousness for longer than 5 minutes
- Repeated vomiting or vomiting for more than 8 hours after injury
- Instability following multiple traumas
- Tense fontanelle in child
- Fracture of skull with any of the following:
  - Suspected open or depressed skull fracture
  - Any sign of basal skull fracture (haemotympanum, 'raccoon' eyes, CSF otorrhoea or rhinorrhoea, Battle's sign)
  - Skull fracture if associated with 1. Impaired consciousnes, 2. Fits, 3. Neurological symptoms or signs

Table 2.11 Information to be looked on CT scan

- Integrity of soft tissue and bone, the size of fontanel and suture lines, and presence of foreign bodies
- Appearance of the normal structures, presence or absence of haemorrhage, & signs of oedema, infarct, or contusion
- Mass effect as indicated by midline shift
- Appearance of the ventricles and cisterns: Compression of the ventricular enlargment may suggest development of hydrocephalus from intraventricular haemorrhage or blockage by mass effect
- Presence of cerebral oedema as indicated by loss of gray-white matter d emarcation

MRI is useful for estimating the initial mechanism and extent of injury and predicting its outcome in neurologically stable patients. MRI is superior to CT scan for identifying diffuse axonal injury (DAI) and small intraparenchymal contusions. This examination is useful to show long term effects of head injury. Magnetic resonance angiography may be used in patients with head injury to assess for arterial injury or venous sinus occlusion.

CHAPTER 3

Management of Head Injuries

PREHOSPITAL TRAUMA CARE

Prehospital care is important and is the start of the trauma chain. Philosophy of prehospital care is to perform basic resuscitation at the scene of accident as up to 30% of prehospital deaths are preventable. At the scene of accident as an initial action, important measures should be performed (Table 3.1) particulary by paramedical staff and physicians can guide them to perform these actions carefully (for example, from ambulance driver).

Table 3.1 Initial actions at the scene of accident

- Make the accident site as safe as possible before assessing any c asualties
- Determine the nature of accident & likely mechanism of injuries
- Determine number, direction and types of vehicles involved
- Degree of intrusion of damaged vehicles
- Whether occupants were wearing seatbelts

PREHOSPITAL RESUSCITATION

Prehospital resuscitation can be carried out by paramedical persons and should follow same principles as that in hospital. Following guidelines will help in prehospital resuscitation and need to be adapted according to circumstances:

- Airway management can be difficult but can be maintained with basic measures (i.e., jaw thrust)

- Avoid intubation without anaesthesia as it can induce vomiting and raised intracranial pressure
- Cervical spine should be immobilized with a hard collar
- Oxygen should be given by mask
- Control haemorrhage with direct pressure
- If patient is entrapped inside, ensure good venous access before releasing from vehicle
- Fluid resuscitation to maintain a systolic blood pressure of 90 mm Hg
- If venous access is difficult, consider rapidly transfering the patient rather than delaying transfer
- Look for potential sign of significant truama (Table 3.2)
- Patient should be packaged for transport and it may require hard collar, head blocks, limb splints

Table 3.2 Indicators of potentially significant trauma

- Penetrating injury to chest and abdomen
- Two or more proximal long bone fractures
- Evidence of high-energy impact
- Fall more than 6 m
- Crash speed greater than 20 mph
- Inward deformity of car of more than 0.6 m
- Rearward displacement of front axle
- Ejection of passenger from vehicle
- Rollover of passenger from vehicle
- Rollover of vehicle
- Death of another car occupant
- Pedestrian hit at greater than 20 mph
- Abnormal physiological variables (i.e., hypotension, tachycardia)
- Burns involving more than 15% of body surface area
- Burns to face and airway

POTENTIAL PROBLEMS AT SCENE OF ACCIDENT

While handling and managing patients at the scene of accident remember following things:

- Assess safety of yourself and patient
- Assess hostility of environment
- There may be lack of familiarity with surroundings
- Intrusive onlookers may disturb the rescue work

EMERGENCY MANAGEMENT OF HEAD INJURIES

The goal of emergency care of patients with head trauma is to recognize and treat life-threatening conditions and to eliminate or minimize the role of secondary brain injury. Patients with severe head trauma are at increased risk of developing cerebral oedema, respiratory failure, and herniation secondary to the increased ICP; therefore, frequent serial assessments of neurological status must be performed. The Brain Trauma Foundation has developed guidelines regarding the medical management of patients with severe head injury. These guidelines suggest that cardiopulmonary resuscitation should be the foundation upon which treatment of intracranial injuries must be based. Initial evaluation of a person who is injured critically from multiple traumas is a challenging task, and every minute can make a difference between life and death.

Patients should be managed according to ATLS protocols (Table 3.3) to prevent secondary brain injury and remember that 5–10% patients with severe head injury may have associated cervical spine injuries.

Table 3.3 ATLS methodology

Primay survey and resuscitation

- A = Airway and cervical spine
- B = Breathing
- C = Circulation and haemorrhage control
- D = Dysfunction of the central nervous system
- E = Exposure

Secondary survey

- Definitive treatment

Full assessment in emergergency requires:

- Pulse, blood pressure
- Glasgow coma scale
- Assessment of pupil diameter and response
- Assessment of limb movement
- Patients with GCS less than 8 require early intubation

Intravenous steroids are of no benefit.

Causes of Mortality (Table 3.4)

Before proceeding we should know the causes of mortality in trauma patients as it will give us an idea how we can avoid trauma-related deaths. Mortality can be grouped into immediate, early, and late deaths and all organized systems for trauma care are focused on the salvage of a patient from early trauma mortality, while critical care is designed to avert late trauma mortality.

Early trauma deaths result from failed oxygenation of the vital organs, massive central nervous system injury, or both. The mechanisms of failed tissue oxygenation include inadequate ventilation, impaired oxygenation, circulatory collapse, and insufficient end-organ perfusion. Massive central nervous system trauma leads to inadequate ventilation and/or disruption of brainstem regulatory centers. Injuries that cause early trauma mortality occur in predictable patterns based on the mechanism of injury; the patient age, sex, and body habitus; or environmental conditions.

Table 3.4 Causes of trauma related mortality

Mortality	Time duration	Causes
Immediate mortality	Within minutes at the scene of injury	Fatal disruption of the great vessels, heart, and lungs or a major disruption of body cavities Major neurological injury Medical treatment can rarely improve outcome
Early mortality	15 minutes to 6 hours 'golden hours'	Cardiovascular and/or pulmonary collapse Intracranial haematoma Major thoracic or abdominal injury Primary focus of intervention for the advanced trauma life support (ATLS) methodology
Late mortality	From days to weeks	Sepsis and multiple organ failure

PRIMARY SURVEY AND RESUSCITATION

The main aim in this stage is prevention of secondary injuries and for that ensure adequate ventilation and perfusion to maintain partial pressure of oxygen > 60 mm Hg, partial pressure of carbon dioxide 30–35 mm Hg, and systolic BP > 90 mm Hg. The level of consciousness can be lowered independent of head injury for numerous reasons in these patients (Table 3.5) and a more reliable and meaningful assessment of severity is provided by the post-resuscitation GCS score, which generally refers to the best level obtained within the first 6–8 hours of injury following nonsurgical resuscitation.

Table 3.5 Causes of lower level of consciousness in head injury

1. Shock	4. Alcohol intoxication
2. Hypoxia	5. Postictal state
3. Hypothermia	6. Administration of sedatives or narcotics

Most of the patients with head injury are restless and this may interfere with further evaluation and management. Look for causes of restlessness in these patients (Box) and do not forget certain points in the management of these patients (Box).

Causes of restlessness

- Check airway for obstruction
- Check for urinary retention
- Check for any tight unremoved clothing
- Check for any tight plasters and dressings

Remember

- Any changes in Glasgow coma scale
- Manage severe head injuries as multiply injured patients
- Stabilize the neck
- Beware of intoxication with alcohol or drugs
- Proper record keeping
- If in doubt, admit

Airway and Ventilation

Airway should be secured as the initial action of resuscitation in any patient with trauma. One should remember that:

- A cervical spine injury should be assumed until proven otherwise
- Oxygen should be delivered at high concentration
- Hypercarbia should be prevented

Airway assessment

While assessing the airway, one should ask following simple questions:

- Whether the patient responds appropriately?
- Whether the airway is patent?
- Whether ventilation is intact?
- Whether the brain is being adequately perfused?

> **Remember** that agitation is often a sign of hypoxia in trauma patients

Airway management

Aims of airway management are:

- To secure an intact airway
- To protect a jeopardized airway
- To provide an airway when none is available

There are conditions where it becomes necessary to provide a secure airway (Box) and this can be achieved with basic, advanced and surgical techniques.

Indications to secure airway

- The airway is compromised
- Oxygenation or ventilation is inadequate
- The airway is unprotected
- The level of consciousness is decreased (GCS < 8)
- The patient is combative and at risk of self-injury
- Risk of airway loss in the immediate future exists (e.g., large-area burns)
- Airway control is desired for therapeutic or diagnostic procedures

Basic measures. Basic measures include:

- Removal of foreign bodies from the mouth and oropharynx
- Suction of secretions and blood
- Chin lift or jaw thrust to secure airway if there is toungue fall
- Placement of oropharyngeal or nasopharyngeal airway
- Administer oxygen at a rate of 10–12 L/min
- Oxygen can be administered via a tight fitting mask with reservoir (e.g., Hudson mask)
- Try to achieve a FiO_2 of 85% or more

Advanced measures. If above measures fail and gag reflex is absent, patient will need endotracheal intubation. While making decision for endotracheal intubation remember:

- If there is no cervical spine fracture, then orotracheal intubation is preferred
- If cervical spine injury cannot be excluded, consider nasotracheal intubation
- The position of the tube after intubation should be checked

Complications of endotracheal intubation include:

- Oesophageal intubation
- Intubation of right main bronchus
- Failure of intubation
- Aspiration

To avoid aspiration, nasogastric tube should be inserted into all patients requiring endotracheal intubation. If a head injury with skull base fracture is suspected or nasotracheal intubation is performed then nasogastric tube should be passed via mouth.

Surgical airways. If unable to intubate the trachea through nose or oral cavity then a surgical airway (an emergency tracheostomy) is indicated. Also, in emergency situation surgical airway can be achieved with a needle or surgical cricothyroidotomy (Box).

Surgical airway
Needle cricothyroidotomy ▪ Cricothyroid membrane is punctured with a 12 or 14 Fr cannula ▪ Connected to oxygen supply via a Y connector ▪ Oxygen supplied at a rate of 15 L/min ▪ Jet insufflation achieved by occlusion of Y connection ▪ Insufflation provided one second on and four seconds off ▪ Jet insufflation can result in significant hypercarbia ▪ Should only be used for 30–40 minutes
Surgical cricothyroidotomy ▪ Small incision made over cricothyroid membrane, 5 mm incision made in membrane and small tracheostomy tube inserted

Complications of surgical airways include:

- Aspiration
- Haemorrhage/haematoma
- Cellulitis
- False passage
- Subglottic stenosis
- Mediastinal emphysema

Ventilation

In the non-intubated patient, ventilation can be achieved either by mouth-to-face mask or bag-valve-face mask (AMBU bag). The latter is more efficient if performed with a two-person technique in that one maintains face seal and the other ventilates the patient. If endotracheal intubation is required it should be performed with cricoid pressure. If patient has rib fractures then insert chest drain on side of injury to prevent pneumothorax.

Blood Pressure Management

In any patient with hypotension and tachycardia (features suggestive of hypovolaemic shock) rule out abdominal injuries, chest injuries and long bone fractures or any obvious source of bleeding as hypotension when present should not solely be attributed to intracranial haemorrhage. Normal saline is the initial fluid of choice for resuscitation in head injury patients that can be supplemented with colloids and blood transfusion. Avoid glucose containing fluids as it will increase the cerebral oedema.

Fluids containing high glucose concentration will harm the brain in two ways:

1. Glucose will get metabolized into water and carbon dioxide and this will decrease the blood osmolality leading to cerebral oedema.
2. At the same time, in ischaemic brain glucose will get metabolized into lactic acid by anaerobic glycolysis and will cause metabolic acidosis that will further injure the neurons.

Remember In a diabetic patient rule out hypoglycaemia and correct it by appropriate measures

Management of Coagulopathy

Disseminated intravascular coagulopathy is present in one-third of head trauma patients and requires aggressive management and correction with replacement factors in order to decrease the risk of further intracranial bleeding and to allow surgical intervention when necessary.

Position of Patient

Elevation of the head to 30° and maintaining midline position continues to be recommended because it improves the venous drainage and decreases the ICP without affecting the CBF (cerebral blood flow). A cervical spine collar should be used until clearance of the spine is achieved.

Seizure Management

Posttraumatic seizures can complicate head injury in 10% of patients and may affect the outcome adversely by increasing the ICP, increasing the metabolic demands of the brain, and causing hypoxia and/or hypoventilation in a spontaneously breathing patient. Adequate control of seizures should be obtained with standard anticonvulsant medication.

Indications for anti-epileptics in acute head trauma

- Severe head injury—GCS < 8
- Head injury with localizing signs

- Depressed skull fracture
- Any case where an intracranial mass lesion has been diagnosed
- Any case of intracerebral contusion

Doses. Load all of the patients at high risk of seizures with phenytoin (diphenylhydantoin) 10 mg–20 mg/kg as IV infusion in 200 mL of saline (**NB**: Phenytoin is not compatible with any glucose containing fluid) over 1/2 hour–1 hour. Infusion should be given slowly to reduce risk of arrhythmias and hypotension, viz., adult < 50 mg/min, children < 1–3 mg/kg/min. In children < 2 years phenobarbitone 10 mg/kg as slow IV injection should be given.

SECONDARY SURVEY AND DEFINITE CARE

After resuscitation, the first step towards management of head injury is to record Glasgow coma score and ensure that it is repeated regularly (every 15 minutes) by nursing staff. Remember that your initial neurological assessment has little value in itself. Its main use is that it provides a baseline to which subsequent scores can be compared. A decrease in the coma score of 2 or more indicates significant deterioration. Along with GCS, pupillary examination and limb movements should also be recorded periodically. This should be supplemented with detail examination of the patient to rule out chest, abdomen, long bone or any systemic injuries.

Criteria for Hospitalization (Table 3.6)

Criteria for hospitalization should be directed on an individual basis. Many accident and emergency departments have an associated observation ward where patients with mild head injuries can be kept for observation. All patients with severe head injuries should be admitted. Patients with severe injuries can be transferred to neurosurgical units where skull fractures, intracranial haematomas and other intracranial injuries can be expertly managed.

Table 3.6 Criteria for admission in head injuries

- Imparied consciousness at the time of assessment or a history of loss of consciousness for more than 5 minutes
- Seizures

Contd.

Table 3.6 Contd.

- Persisting neurological symptoms or signs
- Focal neurologic deficit
- Protracted vomiting, severe and persistent headache
- Difficult assessment: drugs, alcohol or other intoxication
- Skull fracture
- Suspected child abuse
- Unreliable or no responsible caregiver
- Underlying pathology such as coagulopathy or hydrocephalus
- Patients with concomitant diseases or medications that pose increased risk (for example, coagulopathies & anticoagulants)
- ICU admission should be based upon the severity of trauma and associated injuries

Indications for Surgery (Table 3.7)

Decision to operate on a head-injured patient is based on a number of factors including premorbid state (previous medical history and functional ability of the patient), severity of initial injury, onset and rapidity of neurological deterioration and patient assessment on arrival at the neurosurgical unit. Important radiological features on CT scan include size of focal lesion(s) together with any associated surrounding oedema and midline shift. Also to be considered, particularly in the case of elderly dependant patients, are the wishes of relatives. Before embarking on a neurosurgical procedure, it is important to correct any clotting deficiencies and order required amount of cross-matched blood. With the aid of the CT scans, operation is then planned in consultation with consultant neuro-surgeon-on-call.

Table 3.7 Indications for neurosurgical treatment of the head-injured patient

- Intracranial haematomas that cause significant mass effect
- A midline shift of 5 mm or more
- Obliteration of the basal cisterns on the CT scan (obliteration of the basal cisterns is a reliable predictor of the presence of an intracranial mass lesion that requires evacuation)
- ICP > 30 mm Hg (used in conjunction with neruologic examination to determine which patients with intracranial haematomas may require surgery)

Contd.

Table 3.7 Contd.

- Large (> 30 cc) temporal-lobe haematomas (the risk of developing tentorial herniation is much greater with temporal lesions than with frontal or parieto-occipital lesions)
- Posterior fossa haematomas (patients with posterior fossa haematomas can deteriorate rapidly)
- Contusions > 2 cm (these should be evacuated if they are causing significant mass effect)
- Compound depressed fractures
- Skull fractures with CSF leak
- Gunshot wounds: surgical treatment generally involves a reasonable degree of debridement with antibiotic coverage

Exploratory burr holes

In exceptional circumstances, neurosurgical intervention, in the form of exploratory burr holes, may be made at the referring hospital. Indications for exploratory burr holes include:

- Lack of scanning facilities
- Inaccessible remote areas
- Patients unfit for transfer
- Rarely those patients who physically will not fit into the CT scanning machine because of morbid obesity.

Procedure. Exploratory burr holes may be fashioned in consultation with the nearest neurosurgical unit. Following the administration of a general anaesthetic, with the patient supine and head positioned in a horseshoe head rest, the scalp is shaved and prepared with povidone-iodine solution and draped. Three separate approximately one inch-long linear incisions are then made. Their orientation should be so as to allow them to be joined up at the neurosurgical unit to produce a formal flap. These can be located one finger breadth anterior to the coronal suture in the pupillary plane (frontal burr hole), one 2.5 cm anterior to the tragus and 2 cm above the zygomatic arch, on the pterion and the other over the parietal bony eminence (parietal burr hole); repeated on the opposite side if no haematoma is found (Figure 3.1). Using a Hudson-Brace drill, with first the perforator to just penetrate the inner table of the skull, and then to extend the opening with the burr, the underlying dura is exposed. An extradural haematoma may be visible and if present can be removed by suction and then lavaged with sterile saline. Any vessels are diathermised with bipolar forceps and the dura is then opened with a number 11 blade in the cruciate fashion. The dural edges are diathermised with bipolar forceps. An underlying subdural haematoma may be visible and can be decompressed with gravity and gentle saline lavage, mindful of the underlying brain. The neurosurgeon must be informed of the results of such a procedure and the appropriate postoperative care instituted.

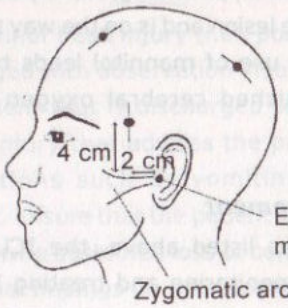


Fig. 3.1 Location for exploratory burr holes

Medical Management

Medical therapies are aimed at providing the injured brain with an adequate cerebral perfusion pressure, avoiding hypoxia and hypercarbia, and decreasing the cerebral oxygen demand. Patients with significant head injuries should be idealllly ventilated to provide adequate oxygenation and ventilation (PCO_2 of 4–4.5 kPa or 35–40 mm Hg).

Hypoventilation (PCO_2 >40 torr) and severe hyperventilation (PCO_2 <30 torr) should be avoided.

Care should be taken to avoid interfering with venous drainage from the face and head by not using tight endotracheal tube ties or ill-fitting cervical collars.

The patient should be rendered at least euvolaemic and should have a mean arterial pressure (MAP) of at least 90 mm Hg, provided the intracranial pressure (ICP) is less than 20 mm Hg. Otherwise, the MAP may need to be higher to maintain a cerebral perfusion pressure (CPP) of 70–90 mm Hg.

Hyperthermia should be avoided, and patients with this condition should be treated aggressively. Although no convincing evidence supports the routine use of moderate hypothermia, it is practiced by many units. The best outcomes appear to be in the younger age group of men with brain injury .

Mannitol (0.5–1 g/kg) should be administered only to a patient with a lateralizing neurologic examination, posturing, rapidly deteriorating and who has a surgically correctable lesion and is on the way to the operating room. Otherwise, the routine use of mannitol leads to intravascular volume depletion and diminished cerebral oxygen delivery from cerebral hypoperfusion.

Intensive care unit management

In addition to those measures listed above, the ICU management focuses more specifically on monitoring and treating ICP elevations, manipulating cerebral perfusion, providing adequate nutrition, and controlling metabolism. In addition, an ongoing assessment of the patient progress can be made through repeated clinical assessments, CT scans, and other hospital or regionally specific advanced monitoring techniques (e.g., somatosensory-evoked potentials, positron emission tomography or single-photon emission computed tomography imaging).

Cerebral perfusion and blood flow

Cerebral perfusion pressure (CPP) can be estimated by subtracting the ICP from the MAP. CPP should remain at greater than 70 mm Hg. Cerebral blood flow (CBF), although difficult to measure, is autoregulated in the uninjured state over a wide range of MAPs. Head injury uncouples this tightly coupled process such that CBF may not match or be adjusted in parallel with cerebral metabolic consumption of oxygen. This phenomenon is most notable immediately surrounding a zone of injury, but it also may affect uninjured segments. ICP monitoring is useful in titrating therapy to maintain a set CPP range. A normal ICP is 15 mm Hg; yet, as much as 20 mm Hg is considered acceptable in a trauma patient. A strong correlation exists between elevations in ICP and a poor cerebral outcome. ICP can be measured with the use of many techniques; however, the criterion standard is considered to be a fluid-coupled ventriculostomy catheter inserted into a lateral ventricle. Other devices may be placed into the brain parenchyma; however, these devices do not afford the ability to remove cerebrospinal fluid (CSF) as a treatment for elevations in ICP. Indications for ICP monitoring in patients with a closed-head injury relate to the inability to obtain a reliable neurologic examination (i.e., coma), to manage acute hydrocephalus, or following an operative procedure for the management of an intra-axial or extra-axial collection.

ADVICE ON DISCHARGE

Patients with minor head injury (i.e., post-resuscitation GCS of 14–15) can be discharged with observation instructions in the care of a reliable adult. Any patient that is discharged should be given an information card on head injury that advises the patient to return if he develops any complications such as vomiting, severe headache, visual disturbance etc. Ensure that the patient will be in the care of a competent adult. Patients who sustained loss of consciousness less than 5 minutes and have normal findings on neurologic examination, no symptoms of increased ICP such as vomiting or headache, no signs of basilar skull fracture, and normal findings on CT scanning or skull radiography can also be discharged with close observation by a reliable adult.

CHAPTER 4

Scalp and Skull Injuries

SCALP INJURIES

Scalp injuries are often observed with brain injuries as scalp injuries can overlie other intracranial pathology; therefore, it requires careful inspection and exploration for foreign bodies or underlying skull fractures.

Anatomy

Scalp consists of five layers; the first three layers are bound together and moved as a unit. These layers are:

S-Skin

C-Subcutaneous tissue. This is a fibrofatty layer. Fibrous septa in this layer unite the skin to the underlying aponeurosis of the fronto-occipitalis muscle. The scalp vessels lie within this layer.

A-Galea aponeurotica or epicranial aponeurosis. This is a thin fibrous sheath attached to the bellies of the fronto-occipitalis muscle. Deep to it is the sub-aponeurotic space, which is continuous with the musculoaponeurotic system of the frontalis, occipitalis, and superficial temporal fascia and underlying loose areolar tissue.

L-Loose areolar layer. Occupies the subaponeurotic space and this layer contains emissary veins.

P-Pericranium. It is periosteum of the skull bone.

Scalp is a very vascular structure and lacerations of scalp if not handled properly can cause severe loss of blood. Blood vessels within the scalp do not constrict when injured because the wall is adherent to the surrounding fibrofatty tissue in subcutaneous area. Bleeding can be controlled by applying pressure or suturing the scalp or averting the scalp on itself. Scalp is susceptible to all types of injury, particularly laceration as it is readily crushed and split against the underlying bone. Such lacerations are often linear due to the convexity of the underlying skull. The scalp often swells markedly due to oedema (waterlogged tissues) or haematoma formation (raised swelling) due to bruising above or below the galeal layer.

Remember Dense hair may mask scalp injuries. Shave the surrounding area while examining these wounds.

Scalp Lacerations

Lacerations of the scalp have characteristic features because of its peculiar anatomy and location. In scalp lacerations associated with compound fractures, the prevention of sepsis leading to meningitis is the principle aim in emergency care.

Scalp lacerations gape open because the intact scalp envelops the skull quite tightly. A severely fractured skull may be malshaped, yielding, or moulding parts.

The subarachnoid space is protected by the skin, galea, and, in certain areas, by temporal and occipital muscles, pericranium, bone, dura, and arachnoid. The deeper the penetration of the wound, the greater chance of meningitis and thus the necessity of alert care in preventing infection.

Subcutaneous layer possesses a rich vascular supply and contains an abundant communication of vessels. They do not retract as do vessels in other areas of the body and can result in a significant blood loss.

The relatively poor fixation of the galea to the underlying periosteum of the skull provides little resistance to shear injuries that can result in large scalp flaps or so-called scalping injuries.

This layer also provides little resistance to haematomas or abscess formation, and extensive fluid collections related to the scalp tend to accumulate in the subgaleal plane.

Caput Succedaneum and Cephalohaematoma

Caput succedaneum and cephalohaematoma are observed with birth-related head trauma. Caput succedaneum involves moulding of the neonatal head and crosses the suture lines, whereas cephalohaematoma involves subperiosteal bleeding and is limited by the suture lines.

Clinical Examination

After managing the life-threatening injuries, a complete examination of the scalp is necessary (Box).

- Inspect the head and the face for abrasions, swelling, ecchymosis, missing tissue, lacerations, and haemorrhage.
- Inspect open wounds for foreign bodies.
- Inspect open wounds for fractures.
- Palpate skull for fractures.

Investigations

Lab studies

- Complete haemogram including haemoglobin and haematocrit when excessive bleeding occurs
- Blood type and cross match
- Wound cultures from lacerations or open skull fractures, when infection is suspected.

Imaging

X-rays. Plain radiographs of skull to rule out any foreign body or fracture.

CT scan. CT imaging is superior to plain films in helping delineate underlying fracture or brain injury.

Management

Severe scalp bleeding can be controlled by a combination of pressure, packing, cauterization, and suturing. Definitive repair of the wounds should be undertaken once the patient's condition has been stabilized.

General principles are to be followed to repair these wounds for best results:

- Margins must be free from undue tension and be everted at the skin edges for good cosmetic healing
- Prevent skin tension by closing the deeper dermis and fatty tissue separately.
- Avoid dead space as it may lead to infection.
- Meticulous use of absorbable and non-absorbable suture material.
- Avoid excessively tight wound closure as it may lead to skin necrosis

To minimize postoperative oedema, a light pressure dressing consisting of gauze and a head wrap may be placed over the operated areas. If the dressing remains dry, it may be removed after 2–5 days. Postoperative antibiotics will be needed if there is infection or gross contamination of the wound covering Gram-positive and anaerobic organisms for 5–10 days.

SKULL FRACTURE

Introduction

The solid skull is deformed by localized impact and if the force and deformation is strong enough it will fracture at or near the site of impact. Skull fractures are called simple if there is no communication between the fracture and the atmosphere and compound if there is such communication. Uncomplicated skull fractures themselves are rarely lethal but associated intracranial damage maybe lethal. A fracture indicates that substantial force has been applied to the head which is likely to have damaged the cranial contents. Skull factures may occur with no associated neurological damage and conversely, fatal injury to membranes, blood vessels and the brain may occur without overlying fracture. Location of the fracture is important because it may cross the path of a major vessel and be associated with an intracranial bleed.

Anatomy

The skull is formed by the fusion of several plates of bone, which are solidly held together along the cranial sutures. Each skull bone consists of a hard and thick outer layer or table of bone, the cancellous middle layer or diploe and the thinner inner table.

The calvarium is covered by periosteum on both outer and inner surfaces. On inner surface, it fuses with the dura to become the outer layer of dura. The average thickness is approximately 5 mm and thickest area is usually the occipital bone and thinnest is the temporal bone.

Pathology

The causative forces and fracture pattern including type (Figure 4.1), extent, and position are important in assessing the sustained injury. Skull is thickened at the glabella, external occipital protuberance, mastoid processes, and external angular process and is joined by 3 arches on either side. Skull vault is composed of cancellous bone (diploe sandwiched between 2 tablets, the lamina externa [1.5 mm], and the lamina interna [0.5 mm]). Diploe does not form where the skull is covered with muscles, leaving the vault thin and prone to fracture. Skull is prone to fracture at certain anatomic sites that include the thin squamous temporal and parietal bones over temples and sphenoid sinus, foramen magnum, petrous temporal ridge, and inner parts of sphenoid wings at the skull base. Middle cranial fossa is the weakest, with thin bones and multiple foramina. Other places prone to fracture include cribriform plate and roof of orbits in anterior cranial fossa and areas between the mastoid and dural sinuses in posterior cranial fossa.

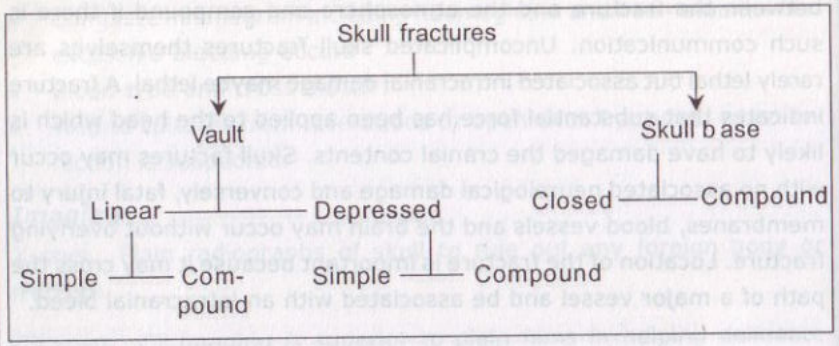


Fig. 4.1 Types of skull fracture

Cranial vault fractures

These are indicative of the severity of the injury, and uncomplicated fractures are not of great significance otherwise, except that temporal bone fractures may predispose to extradural haematoma. These patients may require surgery to elevate the bone fragments. These fractures can be well demonstrated on CT.

Comminuted fractures

Comminution type skull fractures result from a high-energy direct blow to a small surface area of the skull with a blunt object. Comminution of fragments starts from the point of maximum impact and spreads centrifugally.

Linear or fissure fracture

This involves the skull vault and can extend down to the base of skull. Linear fracture indicates that there has been a significant injury to the head. In children, 90% of the fractures are linear and tend to be more diastatic; thus, the radiographic appearance is more impressive. An open linear fracture is a fracture overlaid by a laceration. The presence of cerebrospinal fluid (CSF) in the wound indicates a violation of the dura and warrants further exploration. Linear fracture results from low-energy blunt trauma over a wide surface area of the skull. It runs through the entire thickness of the bone and, by itself, is of little significance except when it runs through a vascular channel, venous sinus groove, or a suture. In these situations, it may cause epidural haematoma, venous sinus thrombosis and occlusion, and sutural diastasis, respectively. Differentiating features between sutures, vascular markings and fractures are summarized in Table 4.1.

Depressed fracture

Depressed skull fracture is defined as displacement of the inner table of the skull by more than one thickness of the bone. If trauma is more severe the skull fragment is circumferentially broken and pushed below the level of the skull. This bone fragment is usually broken into several pieces. This can result in dural tear and laceration of the underlying brain. It is important to point out that skull fragments should be replaced to avoid the creation of skull defect and need for cranioplasty. Underlying dura should be repaired and any bleeding controlled. This is important in children below the age of 4 to avoid complications of growing skull fracture. As mentioned above, bone fragments should be replaced even in compound fractures and wound debrided.

Table 4.1 Differentiating features between fracture, sutures and vascular markings

Fractures	Sutures	Vascular markings
▪ Straight translucent lines	▪ Winding serpiginous lines	▪ Less translucent – affect inner table only
▪ More radiolucent – factures affect both tables	▪ Fine sclerotic or corticated margins	▪ Not sharply demarcated
▪ Most are straight but can change direction suddenly	▪ Typical anatomical sites	▪ Meningeal grooves taper as they run distally
▪ Sharply demarcated	▪ Symmetrical	▪ Branching pattern and symmetrical
▪ Parallel margins – no tapering		▪ Diploetic venous channels are wide
▪ May run across sutures		

Depressed skull fractures result from a high-energy direct blow to a small surface area of the skull with a blunt object such as a baseball bat. Comminution of fragments starts from the point of maximum impact and spreads centrifugally. Most of the depressed fractures are over the fronto-parietal region because the bone is thin and the specific location is prone to an assailant attack. A free piece of bone should be depressed greater than the adjacent inner table of the skull to be of clinical significance and requiring elevation. A depressed fracture may be open or closed, close and contaminated. Open fractures, by definition, have either a skin laceration over the fracture or the fracture runs through the paranasal sinuses and the middle ear structures, resulting in communication between the external environment and the cranial cavity.

Depressed fractures vs subgaleal haematoma

Differentiation between subgaleal haematoma and a depressed fracture is made by evaluating the edges of the lesion. The edges are usually smooth in haematoma and the circumference is rather regular. In depressed fractures, the edges are usually rough, irregular, and sloping. Careful pressure over a haematoma will ordinarily push aside any central indentation; but in fracture, no such shifting of the depression occurs.

Skull base fracture

This starts as vault linear fracture and extends into the skull base. It follows the weak points in the skull as the cribriform plate, foramina and internal ear. Nerve injury can result involving the olfactory nerves. Other cranial nerves could be affected such as the facial nerve. If the fracture extends into the cribriform plate and is associated with dural tear, CSF leak can result and this is called rhinorrhoea. If the fracture extends into the internal ear and the middle ear it can lead to CSF otorrhoea. Other symptoms, i.e., loss of consciousness, seizures, and neurologic deficits may or may not be present. Children with basilar skull fracture usually have prolonged nausea, vomiting, and general malaise, most likely because of the vicinity of the fracture to the emesis and vestibular brainstem centers. These fractures are not always visible, but blood in the sinus cavities (e.g., sphenoid sinus) suggests their presence. This is important as such patients are prone to develop meningitis and require antibiotic prophylaxis. If the patient has clinical evidence of skull base fracture (e.g., CSF rhinorrhoea or bleeding from the external auditory meatus), a normal CT does not exclude such a fracture.

Clinical Features

Linear skull fracture

Most patients with linear skull fractures are asymptomatic and present without loss of consciousness. Swelling occurs at the site of impact, and the skin may or may not be breached.

Depressed skull fracture

Approximately 25% of patients with depressed skull fracture do not report loss of consciousness, and another 25% lose consciousness for

less than an hour. The presentation may vary depending on other associated intracranial injuries such as epidural haematoma, dural tears, and seizures. A thorough examination of local wound is must and look for any CSF leak as depressed bone fragments may cause a laceration of the dura mater (Figure 4.2). The outcome following a depressed skull fracture is based upon the underlying brain injury. If no brain injury is present the surgery represents a cosmetic procedure and outcome is generally quite good.

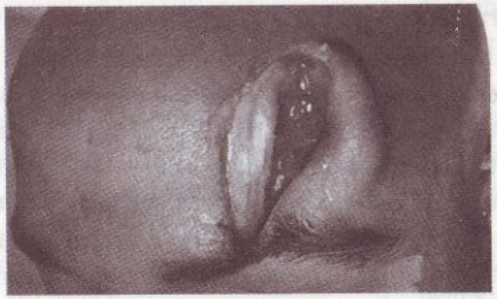


Fig. 4.2 Compound depressed fracture of left frontal bone

Basilar skull fracture

Skull base fractures involve the floor of the skull and include fractures of the cribiform plate, frontal bones, sphenoid bones, temporal bone and occipital bones. The clinical features of a basal skull fracture will depend on site of fracture (Table 4.2) and may include the following:

Deafness. Longitudinal temporal bone fractures result in ossicular chain disruption and conductive deafness of greater than 30 dB that lasts longer than 6–7 weeks. Temporary deafness that resolves in less than 3 weeks is due to haemotympanum and mucosal oedema in the middle ear fossa.

Facial palsy and vestibular dysfunction. Facial palsy, nystagmus, and facial numbness are secondary to involvement of the VII, VI, and V cranial nerves, respectively. Transverse temporal bone fractures involve the VIII cranial nerve and the labyrinth, resulting in nystagmus, ataxia, and permanent neural hearing loss.

Lower cranial nerve involvement. Occipital condylar fracture is a very rare and serious injury. Most of the patients with occipital condylar fracture, especially with type III, are in coma and have other associated cervical spinal injuries. These patients also may present with other lower cranial nerve injuries and hemiplegia or quadriplegia. Other syndromes of lower cranial nerves are:

Vernet syndrome or jugular foramen syndrome is involvement of the IX, X, and XI cranial nerves. Patients present with difficulty in phonation and aspiration and ipsilateral motor paralysis of the vocal cord, soft palate (curtain sign), superior pharyngeal constrictor, sternocleidomastoid, and trapezius.

Collet-Sicard syndrome is occipital condylar fracture with IX, X, XI, and XII cranial nerve involvement.

Table 4.2 Signs of basal skull fracture

- Blood or CSF from ear or nose (otorrhoea or rhinorrhoea)
- Periorbital h aematoma
- Bruising behind the ears (postauricular ecchymoses) (Battle's sign)
- Haemotympanum (blood b ehind the eardrum)
- Bruising around the eyes (periorbital ecchymoses)
- Injury to cranial nerves
  - VII nerve: weakness of the face
  - VIII nerve: loss of hearing
  - Olfactory nerve: loss of smell
  - Optic nerve: vision loss
  - VI nerve: double vision
- Radiological evidence of intracranial air
- Radiological evidence of fluid levels in sinuses

Investigations

Lab studies

In addition to a complete neurological examination, baseline laboratory analyses, and tetanus toxoid (where appropriate, as in open skull fractures), the diagnostic workup for fractures is radiological.

Imaging studies

Although X-ray proof of fracture is important, many fractures are difficult to demonstrate; clinical evidence may be more important. The examiner should carefully palpate the skull and look for small lacerations hidden within the hair.

Radiographs. Skull X-ray referral criteria panel decided that skull films are suboptimal in detecting basilar skull fractures. Hence, other than a fracture at the vertex that might be missed by CT scan and picked up by a plain film skull X-ray is of no benefit. Abnormally lucent (overlapping bony margins) or dense lines (two thicknesses of bone in a focal area) should be sought as both occur with skull fracture. The skull is subject to linear fractures that appear on the film as thin black lines with ragged edges that may run in any direction. They must be differentiated from suture lines, diploic veins, and other blood-vessel grooves (Table 4.1).

Remember that a fracture extending through the distribution of the middle meningeal artery can produce epidural haemorrhage within a few hours. In adolescents and young adults, suture lines are still present and measure less than 3 mm. Potential arterial or venous bleeding or throm-bosis of the dural sinuses may be associated with diastasis of the lambdoidal and sagittal sutures. Comminuted and stellate fractures are generally obvious on radiographs.

A depressed fracture offers an appearance of a white line because of the overlapping margins of the break. Fractures of either the outer or inner table appear as thin black lines or areas of slightly irregular density and structure of the bone. A tomogram may be necessary for detection of this type of fractures. A basilar skull fracture is the most difficult skull fracture to detect, and almost always requires a basal view and frequently it is overlooked. Pneumocephalus and pineal displace-ment are important findings on X-rays. A slight collection of air (pneum-ocephalus) progressing along the meningeal margins is a roentgenographic sign of skull fracture. The air pocket appears on the film as an area of markedly diminished density (usually frontal). The pineal gland, located in the central portion of the brain, is calcified in 60% of adults, and it may calcify as early as 6 years. Displacement of this gland, noted on either A-P or lateral views of the skull, may be the only indicator of a haematoma producing structural shifts within the cranium.

Fig. 4.3 X-ray skull AP view showing linear fracture left parietal bone

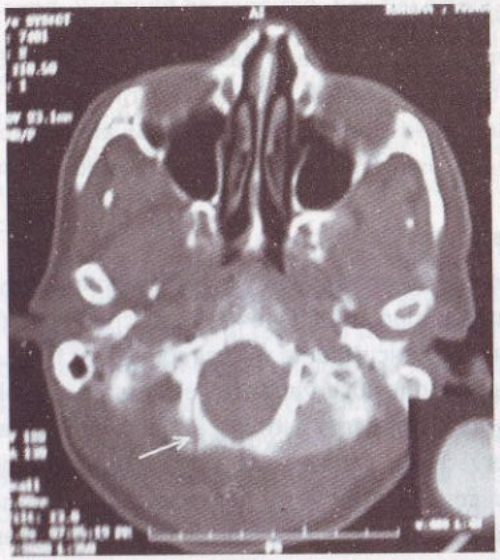


Fig. 4.4 CT scan showing fracture of foramen magnum on right side (type of skull base fracture)

CT scan. CT scan is the criterion standard modality for aiding in the diagnosis of skull fractures (Fig. 4.4, 4.5 & 4.6). Thinly sliced bone windows of up to 1–1.5 mm thick, with sagittal reconstruction, are useful in assessing injuries. Helical CT scan is helpful in occipital condylar fractures, but 3-dimensional reconstruction usually is not necessary.

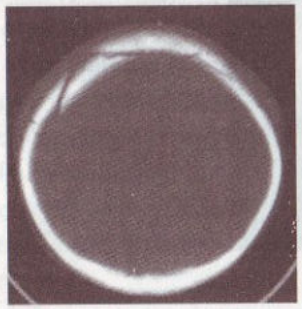


Fig. 4.5 CT scan showing linear fracture right frontal bone

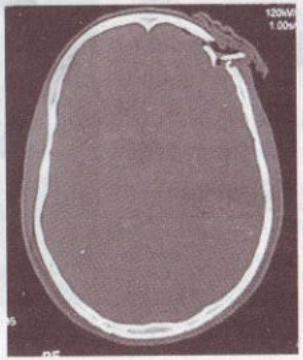


Fig. 4.6 CT scan showing depressed fracture of left frontal bone

Tests for CSF leak

Bleeding from the ear or nose in cases of suspected CSF leak, when dabbed on a tissue paper, will show a clear ring of wet tissue beyond the blood stain, called a halo or ring sign. CSF also can be detected by analyzing the glucose level and by measuring tau-transferrin.

Management

Adults with simple linear fractures who are neurologically intact do not require any intervention and may even be discharged safely and asked to return if symptomatic. Infants with simple linear fractures should be admitted for overnight observation regardless of neurological status. Neurologically intact patients with linear basilar fractures can also be treated conservatively, without antibiotics. Temporal bone fractures are managed conservatively, at least initially, because tympanic membrane rupture usually heals on its own. Simple depressed fractures in neurologically intact infants are treated expectantly. These depressed fractures heal well and smooth out with time, without elevation; however, seizure prophylaxis is recommended for these patients.

Open fractures, if contaminated, will require antibiotics in addition to tetanus toxoid.

Surgical Therapy

The role of surgery is limited in the management of skull fractures. Indications for immediate elevation include gross contamination, dural tear with pneumocephalus, and an underlying haematoma.

Preoperative preparation

Blind probing of skull wounds should be avoided. Patients are prepared for surgery, and exploration is performed in the operating suite under direct vision to prevent loose pieces of bone from damaging the underlying brain. Patients with open contaminated wounds are treated with tetanus toxoid and broad-spectrum antibiotics, especially in a delayed presentation.

Intraoperative consideration

Bony fragments are elevated, and dura is inspected for any tears. If a dural tear is found, it should be repaired. Special attention is given to haemostasis to prevent postoperative epidural collection. Bony fragments are soaked in antibiotic/isotonic sodium chloride solution and are reassembled. Larger pieces may be wired together. Alternatively, titanium mesh also may be used to cover the defect. Methyl methacrylate can be used instead of the bone pieces, but this should be avoided in children.

Dural tear and skull defect. The proper management is to deal with the basic pathology, i.e., the dural tear and the skull defect. The dural edges need to be exposed and this would need removal of bone. The dural edges are then dissected from the underlying parenchyma and repaired in a water tight manner by using some form of dural graft. Following that, the skull defect is repaired by cranioplasty, using autologous bone or metal if the skull defect is too large to be covered with bone.

Venous sinus tears. Depressed fracture over a venous sinus poses a unique situation requiring special attention. The decision to operate is based on the neurological status of the patient, the exact location of

the sinus involved, and the degree of venous flow compromise. A preoperative angiogram with venous flow phase or magnetic resonance angiography is recommended whenever a depressed fracture is thought to be over a venous sinus. Useful data regarding the position and extent of occlusion and transverse sinus dominance is obtained that can affect decisions regarding surgery.

A neurologically stable patient with a closed depressed fracture over a venous sinus should be observed. A patient with an open depressed fracture over a patent venous sinus who is neurologically stable should undergo skin debridement without elevation of the fracture, but if the patient is neurologically unstable, urgent elevation of the depressed fragment is required.

On the other hand, if the patient is neurologically stable and the sinus is thrombosed, it can be assumed that ligation of the sinus will be tolerated. Usually, the anterior one-third of the superior sagittal sinus can be ligated without any consequences; however, tears in the posterior two-thirds need repair, either primarily or with a galea or pericranium patch. Alternatively, a piece of muscle or Gelfoam may be sutured over the sinus.

Skull base fractures. Most basal skull fractures do not require treatment and tend to heal themselves. However, persistent CSF leakage may warrant operative repair in these patients particularly CSF leaks related to frontal bone and cribriform plate fractures. This requires precise detection of the site of leak before any surgical intervention is instituted.

Intracranial air (aerocele, pneumocephalus). The prerequisite for the formation of a typical aerocele is a compound fracture with a ruptured dura (Figure 4.7). This is especially common with a fracture that involves the base of the skull or the sinuses, particularly the frontal sinus or a penetrating injury to the vault (e.g., a bullet wound).

Fig. 4.7 CT scan showing pneumocephalus in bifrontal location

The aerocele is produced by the increased air pressure within the nasal cavity when the patient sneezes or blows the nose. During these events, bacteria may be forced through the fracture into the cranial vault. A combination of symptoms practically pathognomonic for this condition is a history of cranial trauma followed by sneezing, which produces a sudden rhinorrhoea. On X-ray, air may be seen in the subdural space near the fracture line, may fill the subarachnoid spaces and reach the ventricles, or be found within the substance of the brain itself. Symptoms are of slowly increasing intracranial pressure (headache, vomiting and in advanced cases altered sensorium). These patients need antibiotic cover to prevent meningitis. Treatment includes correction of underlying pathology.

Follow-up

Adults with simple linear fractures of the vault, without any loss of consciousness at the time of initial presentation and with no other complications, do not require long-term follow-up. On the other hand, infants with similar fractures with dural tears need to be monitored more closely because of the possibility of the skull fracture expanding. Patients with contaminated open depressed skull fractures treated surgically should be monitored with repeat CT scans a few times over the next 2–3 months to check for abscess formation. Follow-up also is dictated by the complications associated with skull fractures, for ex-

ample, seizures, infections, and removal of bone pieces at the time of initial debridement.

PAEDIATRIC FRACTURES

There are specific conditions that are seen mainly in paediatric age group that needs further discussion.

Ping-pong Fracture

This is a green stick fracture of the skull; it occurs in the first few months of life when the skull bones are still soft. It is caused after falls when the skull hits the edge of a blunt structure such as the edge of a table. It shows as a deformity of the skull, it looks as a shallow trench on the surface of the skull. If large and showing a significant deformity, it can be easily treated by elevating the depressed bone fragment. In newborns, ping-pong depressed fractures are secondary to the baby head impinging against the mother sacral promontory during uterine contractions. The use of forceps also may cause injury to the skull, but this is rare.

Growing Skull Fracture (synonyms: enlarging skull fracture, leptomeningeal cyst)

The basic pathology in these cases is tear of the dura underlying a skull fracture. This could be accidental or operative. The growing skull fractures occur only in paediatric age were there is brain growth, that is to say in children younger than 4 years. The pulsatile brain causes compression of the dural tear, which becomes enlarged, with erosion of the bony edges of the fracture. The result is expansion of the dural tear and enlargement of the skull defect as the skull enlarge, in response to the growing brain. The presence of dural tear is necessary for this to occur. This does not happen with an intact dura, as it expands with the growing brain. There are other associated pathological changes that occur with an enlarging skull fracture; these are cystic degeneration of the underlying parenchyma, enlargement of the ipsilateral ventricle towards the skull defect. Occasionally, there is also CSF collection under the skull defect. The patient presents with a pulsatile swelling, headaches, and occasionally contra lateral neurological deficits. Patient may also develop seizures.

FACIAL AND ORBITAL FRACTURES

Facial and orbital fractures are usually the result of high-speed collisions in vehicles following road traffic accident, sporting accidents and interpersonal violence. The most common facial fractures in sports involve nose, mandible, and supraorbital margins. As these injuries may be associated with multisystem trauma, assessment should follow ATLS principles in these patients and immediate surgical referral is recommended. Bleeding from wounds of the face may be profuse because of rich regional blood vessels and sometimes it is difficult to control. Classification of facial fractures and midface fractures (Le Fort fractures) are shown in Table 4.3 and Table 4.4 respectively.

Table 4.3 Classification of facial fractures

Upper third	Frontal bones
Middle third	Zygoma, nasal bones, and maxilla
Lower third	Mandible and teeth

Table 4.4 Classification of midface fractures

Le Fort 1	Fracture detaching palate and maxillary alveolus
Le Fort 2	Pyramidal fracture through sinus wall laterally and nasal bones medially
Le Fort 3	Fracture through frontozygomatic sutures and orbits detaching facial skeleton from base of skull

Assessment (Table 4.5)

First priority in these patients is to perform a primary survey and attend to ABCs and an assessment of airway patency, breathing, circulation, and gross neurologic function. This is followed by a detailed history; particularly, information regarding the mechanism and magnitude of injury, location, and direction of the impact will help obtaining the diagnosis and management of the patient.

Remember that high-energy trauma may be associated with other concomitant injuries. Progressive facial swelling (pumpkin face) or depressions in the upper cheek may indicate a midface fracture.

In any facial fracture, the mouth and tongue are checked for bleeding. Severe bleeding from the nasopharynx or hypopharynx suggests a fracture that has lacerated vessels near the ethmoid sinus. During first-aid, direct pressure and suctioning may be required to maintain an open airway.

Table 4.5 Assessment of patient with facial trauma

Primary survey	▪ Airway compromise from a fracture or haemorrhage should be identified ▪ Bilateral anterior mandibular fractures may allow tongue to fall back ▪ Orotracheal intubation may be required ▪ Haemorrhage should be reduced with mouth props and epistaxis balloons ▪ Anterior & posterior nasal packing may be required
Secondary survey	▪ Palpate orbital rims, zygomatic arches and mandible to identify fractures ▪ Examine eyes carefully ▪ Reduced eye movement may suggest orbital fracture ▪ Subconjunctival haemorrhage may suggest skull fracture ▪ Proptosis and ophthalmoplegia may suggest retrobulbar haemorrhage ▪ Assess sensation in maxillary branch of trigeminal nerve ▪ Intercanthal distance should be 30–35 mm ▪ Intercanthal distance greater than 35 mm suggests a nasoethmoid fracture ▪ Interpupillary distance should be 55 mm ▪ Intraoral examination ▪ Assessment of occlusion and intraoral haematomas

Ask following questions:

- Has the patient had any visual problems such as double or blurred vision?
  - Suspect orbital wall fracture.
- Do teeth come together normally (normal occlusion)?
  - Suspect mandible or maxilla fracture.
- Is patient able to bite down without pain?
  - Suspect mandible and maxilla fracture.
- Does the patient have areas of numbness or tingling on the face?
  - Suspect injury to trigeminal nerve.
- Does the patient have any bloody or clear-fluid discharge from the nose or ears?
  - Suspect skull base fracture.

Radiology

It may be difficult to obtain X-rays in the acute setting. Close scrutiny of the orbital margins is necessary, with particular attention paid to the normal air space in the maxillary and ethmoid sinuses. Post-trauma soft-tissue effusion often obliterates the inferior and medial orbital margins. Bone fragments may be noted near a fracture site, and old injuries may be evident by ossification consequences of haemorrhage. Useful radiographs views for facial fractures are shown in box.

Radiographic views for facial fractures

- Occipitomental views (15° and 30°) for orbital and zygomatic fractures
- Posteroanterior views of facial bones
- Submentovertex view for zygomatic arch fractures
- Orthopantomogram (OPG) for mandibular fractures
- Reverse Towne's view for condyle neck fractures
- Occlusal films for dentoalveolar fractures

CT Scanning

CT scanning allows complete assessment of facial fractures; can be supplemented with 3-D reconstruction (Figure 4.8). It also allows production of a stereolithograph and a 1:1 resin model from the digital image.

Primary Management of Facial Wounds

Clear the airway and check for signs of head injury. Fractures of more than one area of mandible are likely to produce upper airway obstruction

as tongue may fall backward or blood may gather in the hypopharynx. The patient is placed in the semi-prone position to allow drainage of the airway. Prevent or treat for shock, which is always a danger. If the patient is conscious he may sit on the ground with knees drawn up and with his head resting on his arms folded across his knees.

Fig. 4.8 CT scan showing fracture of left orbital roof

Except for minor wounds, surgical consultation should be sought immediately. Digital pressure can be applied to appropriate pressure points to control haemorrhage. In minor abrasions of the face or skull, bleeding can be stopped with cold compression, and the wound can be washed with a tepid saline solution. In more serious cases, a layer of foam under pressure pad may be necessary. Wound can be dusted with an antibacterial powder. A sterile pressure dressing and cold pack can be applied over skin wounds, but never place a dressing within the mouth.

Principles of Treatment

Primary repair of facial wounds and reduction of facial fractures produces the best cosmetic results but it may be delayed for 2 or 3 days if multidisciplinary approach is required. For facial and mandibular fractures open reduction and internal fixation is treatment of choice as it will allow:

- Anatomical reduction of fractures
- Stable internal fixation
- Early jaw mobilization

Specific Considerations

Zygomatic and trimalar fractures. Fractures of the zygomatic arch usually result from a direct blow to the cheek, resulting in mechanical impingement on the coronoid process of the mandible. There may be severe swelling and trismus in attempt to open the mouth. The sunken cheek becomes apparent only after swelling subsides. Cheek trauma may cause trimalar fractures presenting fracture lines through the infraorbital and lateral orbital rim or the zygomatic arch. Displacement depends on the direction of force. Again, early swelling obliterates displacement. Eye injury, diplopia, and infraorbital anaesthesia are common complications.

Orbital blow-out fractures. Blunt trauma to the eye may result in a hydrostatic blow-out fracture of the egg-shell-thin margins of the orbital floor, altering the upper maxillary sinus margin and bulging soft tissues through the orbital floor. The dense orbital rim is usually intact. These fractures often result from a direct blow to the eye by an elbow, knee, fist, ball, or some other blunt object. In evaluating the lower orbital margin in roentgenography, the overlapping anterior rim of the orbit and the deeply seated posterior-inferior rim of the orbit must be located. In most cases of fracture, a third line may be seen, representing a bony fragment. A soft-tissue bulging of periorbital tissue may be the sole indication of a fragment hinging laterally or medially. In doubtful cases, tomography is helpful. Trauma to the infraorbital nerve results in anaesthesia of the cheek. Enophthalmos and diplopia result from displacement of extraocular muscle and fat or supplying nerve entrapment within fracture fragments.

Jaw Fracture. A fractured mandible (often multiple) is a common facial fracture, second only to nasal fracture. Consultation with an orthopaedic or dental surgeon is recommended. A mandible with an impacted fracture heals slowly compared to that of long-bone fractures. Symptoms may include abnormal closure of teeth, inability to swallow or talk, point tenderness, abnormal palpable bony motion, abnormal deviation of the jaw upon opening, bleeding and drooling from the mouth, and ear pain (especially in condyle fracture). In case of fracture of both

jaws, especially, the soft tissues may drop back into the throat and strangle the patient, requiring early tracheotomy. The most frequent and most overlooked fracture site is at the condyle. The upper airway may become obstructed by blood, mucus, or foreign matter causing respiratory failure and death. Maintenance of an open airway and control of haemorrhage are the emergency procedures taking priority.

CHAPTER 5

Intracranial Haematomas

In a patient with reduced level of consciousness, focal deficits or presence of a skull fracture (Table 5.1) suspect intracranial haematomas. In following sections we will discuss the salient features of these lesions and their management.

Table 5.1 Risk of developing intracranial haematoma in a patient with or without skull fracture

GCS	Risk	Other features	Risk
15	1:3,615	None	1:31,300
		Post-traumatic amnesia	1:6,700
		Skull fracture	1:81
		Skull fracture + PTA	1:29
9-14	1:51	No fracture	1:180
		Skull fracture	1:27
3-8	1:7	No fracture	1:27
		Skull fracture	1:4

EXTRADURAL HAEMATOMA

Introduction

An extradural haematoma (synonym: epidural haematoma [EDH]) is a collection of blood in the potential space between the dura and the cranium. Blunt trauma is the most common cause of EDH and as many

as 10–20% of all patients with head injuries are estimated to have EDH. The trauma frequently is a blunt impact to the head from an assault, fall, or other accident. EDHs usually occur in young adults, and are rare before 2 years of age or after 60 years as in persons over 40 years of age dura becomes increasingly adherent to the skull. Males outnumber females 4 to 1. Patients younger than 5 years and older than 55 years have an increased mortality rate.

Pathology

EDH results from interruption of dural vessels, including branches of the middle meningeal arteries, veins, dural venous sinuses, and skull vessels commonly associated with calvarial fractures. Continued bleeding and growth can result in intracranial hypertension. In the posterior fossa, disruption of dural venous sinuses (e.g., transverse or sigmoid sinus) by fracture may lead to EDH. Disruption of the superior sagittal sinus may cause vertex EDH. Epidural haematomas typically result from direct trauma in which a skull fracture causes tearing of an underlying epidural vessel. This focal trauma results in arterial bleeding stripping the dura off the inner skull table to form a haematoma. This accumulation can be immediate or delayed and in a minority of cases is associated with underlying brain injures or brain injuries on the other side of the head. As it is an arterial bleed the clot can get to a significant size within a short period of time with rapid rise in the intracranial pressure and it may be associated with overlying fracture in nearly all (95%) adults and most (75%) children.

Clinical Features

Most epidural haematomas are traumatic in origin, often involving a blunt impact to the head and can be easily overlooked, as mild concussion is followed by a lucid interval before neurological symptoms. Depending on the force of impact, patients may present with no loss of consciousness, brief loss of consciousness, or prolonged loss of consciousness. Only 10–27% of patients have classical presentation of lucid interval. Initially, the concussive head injury force results in an alteration of consciousness. After recovering consciousness, the EDH continues to expand until the mass effect of the haemorrhage itself results in an increased intracranial pressure, a decreased level of consciousness, and a possible herniation syndrome. This lucid interval of recovery may last from a few hours to 2 days. Other presentations include headache, vomiting and seizure, progressive loss of consciousness. Patients may have external evidence of head injuries such as scalp lacerations, cephalohaematoma, or contusions. Systemic injuries may also be present.

Neurological assessment includes level of consciousness, motor activity, eye opening, verbal output, pupillary size and reactivity, and any lateralizing signs such as hemiparesis or plegia. In awake patients with a mass lesion, the pronator drift phenomenon might help to assess [illegible] significance. Immediate surgical intervention is required. With severe intracranial hypertension, a Cushing response may occur. The classic Cushing triad involves systemic hypertension, bradycardia, and respiratory depression. This response usually occurs when cerebral perfusion is compromised by increased intracranial pressure. Antihypertensive therapy during this time may lead to cerebral ischaemia and is counterproductive. Immediate evacuation of the mass lesion alleviates the Cushing response.

Imaging

Skull X-rays

Skull radiographs often reveal a fracture crossing the vascular shadow of the middle meningeal artery branches. An occipital, frontal, or vertex fracture might also be observed. Presence of a fracture does not necessarily guarantee the existence of EDH. However, more than 90% of cases of EDH are associated with skull fractures. In children, this incidence is less because of greater skull deformability.

CT scan (Fig. 5.1)

CT scan is the most accurate and sensitive method of diagnosing acute EDH. The CT findings are characteristic as the space occupied by EDH is limited by the adherence of dura to the inner table of the skull, especially at the suture lines, contributing to the lenticular or biconvex appearance. Characterisitics of EDH on CT scan include:

- EDH forms an extraaxial, smoothly marginated, lenticular, or biconvex homogenous density.
- EDH rarely crosses the suture line because the dura is attached more firmly to the skull at sutures.

- Focal isodense or hypodense zones within EDH indicate active bleeding. Irregular hypodense swirling indicates active bleeding in the majority of patients.
- Air in acute EDH suggests fracture of sinuses or mastoid air cells.

Cerebrospinal fluid (CSF) is not commonly mixed with epidural haematomas; therefore, the haematoma is denser and homogeneous. The quantity of haemoglobin in the haematoma determines the amount of radiation absorbed. The signal density of the haematoma compared to the brain parenchyma changes over time after the injury. The acute phase is hyperdense, i.e., bright signal, on CT scan. The haematoma then becomes isodense from 2–4 weeks, and it becomes hypodense, i.e., dark signal, thereafter. Hyperacute blood may be observed as low-density areas, possibly indicating ongoing haemorrhage. EDH in the vertex region is less common and may be difficult to diagnose on CT scans because of the anatomical location. Vertex epidural haematomas can be mistaken as artifact in traditional axial CT scan sections. Even when correctly detected, the volume and the mass effect may easily be underestimated. In some cases, coronal CT scan sections can be used to evaluate the haematoma on coronal planes.

Remember: Approximately 10–50% of cases of EDH are associated with other intracranial lesions. These lesions include subdural haematomas, cerebral contusions, and intracerebral haematomas.

Fig. 5.1 Characteristic CT scan of extradural haematoma

Cervical Spine Evaluation

Cervical spine evaluation usually is necessary because of the risk of neck injury associated with EDH.

Principles of Management

An extradural haematoma is a surgical emergency and prompt operation is needed. If untreated, there is a high rate of morbidity and mortality but effective and early treatment can result in complete recovery. In cases where there is no diffuse brain injury and the injury is localized to th ea where the fracture and the haematoma is small, prognosis i ellent. In most cases this is an acute condition; however, casionally the bleeding is a result of venous tear and the blood clot develops slowly. This is particularly the case in the frontal and occipital regions. The clot is evacuated through a craniotomy but in acute situations where there are no facilities for major neurosurgical procedure, a burr hole should be done to release the intracranial clot and to reduce the intracranial pressure.

Medical therapy

The treatment of the epidural haematoma depends on a variety of factors. The adverse effect on brain tissue is mainly from mass effect causing structural distortion, life-threatening brain herniation, and increased intracranial pressure.

When managing these patients, there are two optional routes of treatment: immediate surgical intervention and initial, conservative, close clinical observation with possible delayed evacuation. Note that EDHs tend to expand in volume more rapidly than subdural haematomas, and patients require very close observation if treated conservatively. Not all cases of acute EDH require immediate surgical evacuation. If a lesion is small and the patient is in good neurological condition, it is reasonable to observe the patient with frequent neurological examinations. Early follow-up scanning assesses for further increase in haematoma size prior to deterioration. If a rapid size increase is noted, then surgery is indicated. In managing cases of spontaneous EDH, the underlying primary disease process must be addressed in addition to the fundamental principles discussed above.

Surgical therapy

Primary management of extradural haematoma is surgical evacuation and the objectives of surgery are:

- Clot removal to lower intracranial pressure and eliminate direct pressure on the brain
- To stop ongoing bleeding
- To prevent reaccumulation of the haematoma.

EDH with volume greater than 30 mL, thickness of 15 mm, and a midline shift beyond 5 mm tends to require surgical evacuation because most patients with such an EDH experience a worsening of the conscious state and/or exhibit lateralizing signs. Location also is an important factor in the surgical decision. Temporal haematomas, if they are large or expanding, may lead to uncal herniation and more rapid deterioration. EDH in the posterior fossa, which often is related to the interruption of the lateral venous sinus, often requires prompt evacuation because of the limited space available compared to the supratentorial compartment. A blind exploratory burr hole by an inexperienced operator is rarely helpful. Even if haematoma is found it may not be fully evacuated. However, in certain situations it may be lifesaving (Box).

Indications for exploratory burr holes

- Patient is herniating
- Neurosurgeon is unavailable
- Air or ground medical transport is prolonged

Burr hole procedure. Details of exploratory burr holes are discussed in Chapter 3; however, few points need further consideration. Drill hole adjacent to, but not over, skull fracture or in the area located by CT scan. In the absence of CT scan, place a burr hole on the side of the dilated pupil, 2 finger widths anterior to tragus of ear and 3 finger widths above.

Complications

Brain herniation

Many of the complications from EDH occur when the pressure they exert results in significant brain shifting resulting in brain herniation.

Expanding temporal EDH can result in uncal herniation resulting in occlusion of the anterior and posterior cerebral arteries, leading to cerebral infarction.

Duret haemorrhages

Downward herniation of the brain stem can result in Duret haemorrhages within the brainstem, mostly in the pons.

Transtentorial herniation

Transtentorial herniation may result in an ipsilateral third cranial nerve palsy, which often takes many months to resolve once the pressure is relieved. Third cranial nerve palsy is manifested by ptosis, pupillary dilation, and the inability to move the eye in medial, upward, and downward directions.

Prognosis—Mortality/Morbidity

Mortality rates are approximately 0% for patients not in coma preoperatively, 9% for obtunded patients, and 20% for patients in deep coma. Prognosis is excellent if haematoma is drained early and no secondary brain injury has occurred. Clinical awareness and early diagnosis are the keys to successful management. Normal pupillary reactivity prior to surgery is associated with a favourable outcome in 84–100% of patients. When both pupils are dilated, however, a poor outcome or death occurs in the great majority of individuals. Factors associated with higher mortality are shown in box.

Risk factors for higher mortality

- Advanced age
- Associated intradural lesions
- Temporal location
- Increased haematoma volume
- Pupillary abnormalities
- Rapid clinical progression
- Increased intracranial pressure (ICP)
- Decreased Glasgow coma scale (GCS)
- Absence of a lucid interval

SUBDURAL HAEMATOMA

Subdural haematoma is a collection of blood between the dura and arachnoid. Subdural haematomas (SDH) can be classified by the duration of time that has elapsed from the inciting event to the diagnosis and appearance on CT scan into acute, subacute and chronic type (Table 5.1).

ACUTE SUBDURAL HAEMATOMA

An acute SDH is commonly associated with extensive primary brain injuries and occur in 5–25% of patients with severe head injuries. These haematomas are usually a part of severe and diffuse brain injury and associated with significant morbidity and mortality.

Table 5.1 Types of subdural haematoma

Type	Duration	CT appearance
Acute subdural haematoma	Less than 3 days	Hyperdense (white on CT)
Subacute s ubdural haematoma	4–21 days	Isodense (similar to brain on CT)
Chronic subdural haematoma	More than 21 days	Hypodense (dark on CT)

Pathology

The mechanism to produce an acute SDH is high-speed impact to the skull. This causes brain tissue to accelerate relative to a fixed dural structure, which, in turn, tears bridging veins between the surface of the cortex and the dural sinuses. This mechanism also leads to associated contusions, brain oedema, and diffuse axonal injury. Alternatively, a cortical vessel can be damaged by direct cortical laceration. An acute SDH due to a ruptured cortical artery may be associated with only minor head injury, and no cerebral contusions may be associated. Blood accumulates moderately rapidly and steadily expanding mass compresses brain with same secondary effects as from an epidural haematoma. Subdural haemorrhage is confined unilaterally as the dura is firmly fixed to the falx between the hemispheres.

Clinical Features

The average age of a trauma patient without an acute SDH is 26 years, while the average age of patients with an acute SDH is 41 years. The older patients appear to be at greater risk for developing an acute SDH after head injury. This is believed to be due to older patients having more atrophy, which allows more sheer force against bridging veins immediately after impact. The clinical presentation of an acute SDH depends on the size of the haematoma and the degree of any associated parenchymal brain injury. Subdural bleeding usually does not show lucid interval. Traumatic acute SDHs are often associated with more severe generalized brain injury and have poor initial neurologic conditions (low GCS) on admission. Majority of the patients are drowsy or comatosed. Arousable patient may complain of headaches, poor concentration, confusion and motor deficits (e.g., hemiparesis). Common neurological findings in acute SDH and lateralizing signs (as seen in EDH) include:

- *Pupillary asymmetry.* Inequality of pupils (anisocoria) is an important localizing sign, and Hutchinson pupil from compression of the 3rd cranial nerve against the free edge of the tentorium and this unilateral dilated pupil does not react to light. However, in 5–10% cases dilated pupil is contralateral and bilateral in about 1/3 of cases, compared to 3% in extradural haematomas. Bilateral widely dilated and fixed pupils indicate poor prognosis and suggest brainstem death. Thus, on-scene frequent examinations of the pupils are necessary.
- *Hemiparesis.* Pateint may have hemiparesis contralateral to the haematoma due to compression of underlying brain and less commonly, the hemiparesis may be ipsilateral if caused by direct parenchymal injury to the opposite side of cortex or by compression of the cerebral peduncle (contralateral to the haematoma) against the edge of the tentorium cerebelli (Kernohan notch).
- *Less common findings.* Less common findings include papilloedma and unilateral or bilateral cranial nerve VI palsy.

Diagnosis

SDHs are best diagnosed by CT scan. They appear as a crescent shaped mass of increased density over the brain surface. The blood is seen as high attenuation, but may spread more widely in the subdural space,

with a more irregular inner margin. CT scan will also show any associated injuries (i.e., fracture, contusion etc.).

Treatment

Rapid surgical evacuation is generally indicated for patients with SDHs that are greater than 1 cm at the thickest point. Smaller SDHs may not require surgery. A large decompressive craniotomy is often required to evacuate the thick blood clot and to gain access to possible bleeding sites. Cerebral contusions underlying the SDH are often also removed at the same surgery. These patients will also need anti-oedema measures (mannitol, hyperventilation) and a cover of anticonvulsants (phenytoin sodium).

Outcome

The mortality for patients with an acute SDH ranges from 50–90%. A significant percentage of this mortality is from the underlying brain injury and subsequent raised intracranial pressure that develops in the days after injury. Approximately 20–30% of patients will make a functional neurological recovery. Postoperative seizures are relatively common in these patients. In general, a favourable (functional) outcome is more likely in those patients who are treated very soon after injury, those who are younger adults, those with a higher GCS (above GCS of 6 or 7), those with reactive pupils, those without multiple cerebral contusions and those who do not develop difficult-to-control raised intracranial pressure.

CHRONIC SUBDURAL HAEMATOMAS

Introduction

Chronic SDHs are more common in men with a male-to-female ratio of 2:1. Most adults with chronic SDH are older than 50 years with an average age of 68 years. One-quarter to one-half of patients with chronic SDH have no identifiable history of head trauma and in remaining patients it is usually trivial trauma. The average time between head trauma and diagnosis is 4–5 weeks. Risk factors for a chronic SDH include chronic alcoholism, epilepsy, coagulopathy, arachnoid cysts, anticoagulant therapy (including aspirin), cardiovascular disease (hypertension, arteriosclerosis), thrombocytopenia, and diabetes. In younger patients, alcoholism, thrombocytopenia, coagulation disorders, and oral anticoagulant therapy are more prevalent and in older patients, cardiovascular disease and arterial hypertension are more prevalent.

Pathophysiology

Most chronic SDHs are believed to be derived from subdural granulomas (SDGs). The presence of brain atrophy or loss of brain tissue due to any cause, such as old age, alcoholism, or stroke, provides a potential space between the dura and the brain surface for a SDG to form. SDG may occur after head trauma and frequently is asympto-matic. An SDG begins as a separation in the dura-arachnoid interface, which then is filled by cerebrospinal fluid (CSF). Dural border cells proliferate around this CSF collection to produce a neomembrane. Then, fragile new vessels grow into the membrane. These vessels can rupture and become the source of blood into the space, which results in the growth of chronic SDH. The minority of chronic SDH cases are derived from acute SDH cases that have matured for lack of treatment. Chronic SDHs that form acute SDHs have membranes between the dura and haematoma at 1 week and between the brain and haematoma at 3 weeks. As stated above, new fragile vessels grow into these membranes. The haematoma liquefies at 1–3 weeks of age and becomes hypodense on CT scan. If not resorbed, the vessels in the membranes surrounding the haematoma can haemorrhage repeatedly, resulting in the enlargement of the haematoma. Coagulation and fibrinolysis systems are excessively activated in chronic SDH. This results in defective clot formation and recurrent haemorrhage. Chronic SDHs are not usually associated with skull fracture and usually located over dorsolateral surface of frontal and parietal lobes, usually cover the entire surface of the hemisphere. Rarely these are located in other locations and may be bilateral in 15–20% of cases. A small, self-limiting SDH may remain asymptomatic and be an incidental finding at autopsy.

Clinical Features (See Box)

History is stretching to weeks or months and the most common complaint is headache, seen in up to 80% of individuals. Other features like drowsiness, inattentiveness or incoherence of thought are more pronounced than focal neurological signs such as hemiparesis, making the diagnosis more difficult. An erroneous admission diagnosis occurs

in up to 40% of cases and can be confused with stroke, brain tumour, drug intoxication, depression, and senile or other dementias. Consider diagnosis of chronic SDHs in rapid onset of dementia, particularly if it is associated with headache.

Remember Patient may present with spells of hemiparesis or aphasia lasting minutes and indistinguishable from transient ischaemic attacks (TIA)

Chronic SDHs may gradually expand and behave clinically as a tumour.

Chronic SDH clinical features

- Headache
- Slowed thinking
- Confusion
- Changes in personality
- +/– mild hemiparesis
- Fluctuation in the level of consciousness
- Seizures (rare)

Diagnosis

CT scan *(Fig. 5.2)*

Low-density or isodense concavo-convex mass over convexity of the hemisphere 2–6 weeks after the initial bleed, may show only a shift of the midline structures. Bilateral chronic haematomas are often missed because of the absence of lateral shifts. Isodense collections may be better demonstrated after intravenous contrast as the density will then be less than that of the brain. However, this is rarely a problem with more modern scanners.

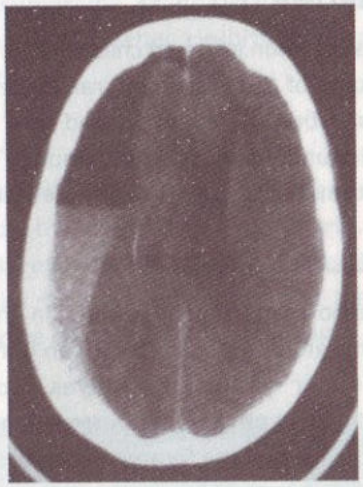


Fig. 5.2 Characteristic CT scan of chronic subdural haematoma

MRI

Demonstrates clot reliably and the intensity pattern varies with the age of haematoma.

Treatment

Patients with symptomatic chronic subdural haematomas are effectively and safely treated with twist drill holes or burr holes and drainage through a catheter passed into the subdural space. This procedure can often be performed under local anaesthesia. Craniotomy is rarely needed. (In contrast, patients with an acute subdural haematoma require a craniotomy for evacuation because the haematoma is clotted and cannot be drained via burr holes.) Sometimes, pseudomembranes may grow from the dura and encapsulate the region, requiring surgical resection to prevent recurrent fluid accumulation. Small haematomas are largely resorbed and only organizing membrane remains, which may become calcified in time.

Outcome

Overall, 80–90% of patients have significant neurological improvement after drainage of a chronic SDH. Residual subdural fluid collections after treatment are common, but clinical improvement does not require complete resolution of the fluid collection.

SUBDURAL HYGROMA (Fig. 5.3)

Subdural hygroma is common in young children and results from arachnoidal tear, with leakage of CSF into subdural space, without significant haemorrhage. It may become encapsulated like a haema-toma and behave like a chronic subdural haematoma, though usually less serious. Treatment of symptomatic lesions is surgical evacuation.

INTRAVENTRICULAR HAEMORRHAGES

Intraventricular haemorrhage tends to occur in the presence of very severe TBI and is, therefore, associated with an unfavorable prognosis. If it results from minor trauma it resolves spontaneously. Large haemorrhages could lead to obstructive hydrocephalus, especially when they are located at the level of the foramen of Monroe and the aqueduct of Sylvius, in which case surgical intervention (i.e., external ventricular drainage) is required.

Fig. 5.3 CT scan showing bilateral frontal subdural hygroma

SUBARACHNOID HAEMORRHAGES

This is the most common form of haemorrhage associated with head trauma and usually results from disruption of the small vessels on the cerebral cortex. In approximately 25–40% of individuals who sustain a moderate or severe head injury (GCS 3–12), subarachnoid haemorrhage (SAH) is visualized on the initial CT scan. The usual location is along the falx cerebri or tentorium and the outer cortical surface. Common symptoms include nausea, vomiting, headache, restlessness, fever, and nuchal rigidity caused by blood in the subarachnoid space.

CT Scan (Fig. 5.4)

Subarachnoid haemorrhages that occur because of trauma are typically located over gyri on the convexity of the brain. The subarachnoid haemorrhages that result from a ruptured cerebral aneurysm are usually located in the subarachnoid cisterns at the base of the brain. Traumatic SAH may be associated with contusion or laceration to the brain surface.

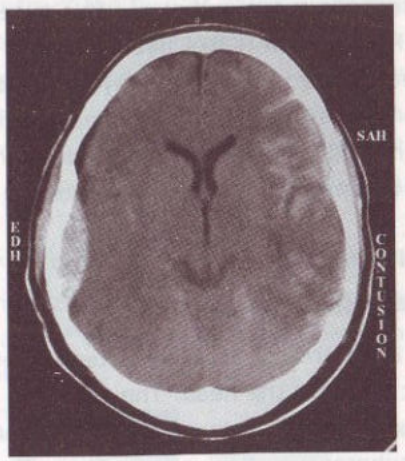


Fig. 5.4 CT scan showing traumatic SAH on left side and thin EDH on right side

Treatment

Treatment of traumatic SAH is usually symptomatic and if it is associated with significant haematoma or contusion then surgical intervention is needed.

Complications

Vasospasm

Vasospasm can follow traumatic SAH and if untreated, the blood supply to a given area of the brain may fall so low that the brain tissue dies, resulting in a stroke. The best way to prevent vasospasm from occurring

or to minimize its impact, is by maintaining an adequate or slightly elevated fluid status (hypervolaemia) of the patient and a normal or even elevated blood pressure.

Communicating hydrocephalus

Traumatic subarachnoid haemorrhage also may lead to a communicating hydrocephalus if blood products obstruct the arachnoid villi or in the event of a noncommunicating hydrocephalus secondary to a blood clot obstructing the third or fourth ventricle.

Outcome

If there is no associated brain pathology, this type of haemorrhage could be benign. However, SAH has been found to be an important factor in severe head injury victims that may be associated with poorer neurological outcomes. Probably the major reason that SAH adversely affects patients with head injury is that it may induce arterial vasospasm, or narrowing of the arteries that supply blood to the brain.

CEREBRAL CONTUSIONS AND INTRACEREBRAL HAEMATOMAS

Cerbral contusion is an area of bruising or tearing of the brain tissue caused by a direct injury to the head. The temporal and frontal lobes are the most vulnerable areas because of their anatomical relationship with the bony protuberances of calvarium. These bruises of the brain may occur without other types of brain haemorrhage or they may be seen with acute subdural or epidural haematomas. These occur due to stretching and shearing injury, often due to impaction of the brain against the skull on the side opposite to the injury. They may be seen directly underneath the site of impact (coup contusion) or diagonally opposite the impact site (contrecoup contusion). When the pia-arachnoid layer is torn, the injury is termed a cerebral laceration. Laceration usually is prominent, with significant extravasation of blood, both subarachnoid and intracerebral.

Multiple petechial haemorrhages (multifocal haemorrhagic contusion)

Multiple contusions may be present throughout the cerebral hemispheres. They are often very small and visible at the grey/white matter interface. They are due to a shearing injury with rupture of small intracerebral vessels, and in a comatose patient with no other obvious cause they imply a severe diffuse brain injury with a poor prognosis. Larger haemorrhages may occur in severe trauma, and they may not be apparent on a scan performed immediately after the injury and a delay is necessary to detect these lesions, usually a day or two after trauma.

Clinical Features

Most patients with cerebral contusions have had a serious head injury with a loss of consciousness. In cerebral contusion or laceration, weakness or paralysis of the face or extremities appears immediately after injury opposite to the side of lesion. This weakness well established at the time of injuy is an important diferentiating point from extradural haematoma where the weakness is progressive. There may be progressive neurologic deterioration secondary to local cerebral oedema, infarcts, and/or late-developing haematomas.

CT Scan (Fig. 5.5)

Cerebral contusions are readily identifiable on computed tomography (CT) scans, but may not be evident on day one scans, only becoming visible at days 2 or 3.

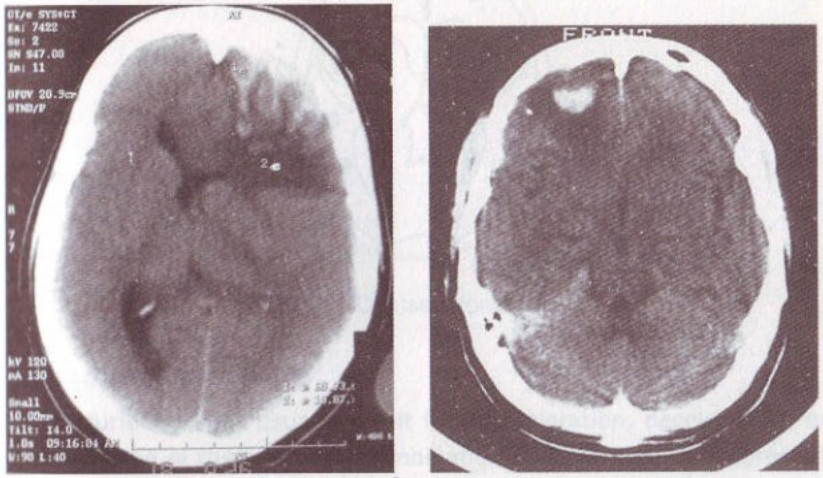


Fig. 5.5 CT scan showing left frontal contusion (right) and right frontal intracerebral haematoma (left)

Cerebral contusions on CT scan appear as irregular regions, in which high-density changes (i.e., blood) and low-density changes (i.e., oedema) are present. Frequently, one of these two types of changes predominates within a particular contusion. Often this haemorrhage is wedge-shaped, apex directed into white matter. CSF contains blood, and oedema around the lesion may be marked.

Treatment

Typically, cerebral oedema (swelling) develops around contusions within 48 to 72 hours after injury. If there is considerable pressure effect (causing raised intracranial pressure) or if the haemorrhage progresses to form a sizeable blood clot in the brain (an intracerebral haematoma), such cerebral contusions may require surgical removal through a craniotomy. Anti-oedema measures and cover of anticonvulsants is required in these patients.

Outcome

Neurological outcome depends on the size and location of contusion in the brain. Other important factors affecting outcome include age, the Glasgow Coma Scale, the presence of other types of intracranial injuries.

CHAPTER 6

Diffuse Injuries

CONCUSSION

Brain concussion is the most common injury to the brain following a cranial blow. It is defined as an essentially transient state due to head injury that has an instant onset, manifests widespread purely paralytic (flaccid) symptoms without neurologic evidence of gross brain injury, and is always followed by a degree of transient unconsciousness and amnesia for the actual moment of the accident.

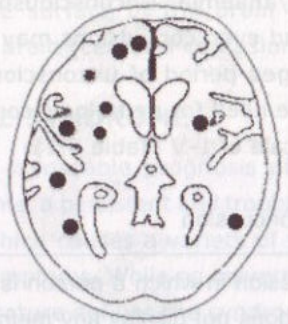


Fig. 6.1 Diffuse axonal injury

Pathophysiology

The injurious forces usually result from acceleration, deceleration, or compression of the head or a combination of these factors. At present it is thought by some to be a manifestation of widespread altered nerve cell metabolism affecting neuronal excitability, resulting from diffuse agitation of nerve cells (molecular commotion).

Other experiments implicate the reticular formation as the major system whose function is abolished in concussion. It is not clearly known why individuals become disoriented or briefly unconscious after a mild head injury. However, in experimental animal models of concussion, the brain injury triggers a brief period of increased neurotransmitter release and elevated brain metabolism. This is followed by a period of depressed brain metabolism which then gradually returns to normal after several days. It is postulated that similar metabolic changes occur after human concussion. Associated cerebral oedema and congestion can cause a moderate rise in cerebral venous pressure.

Clinical Features

Concussion is characterized by loss of consciousness and post-traumatic amnesia. Duration of post-traumatic amnesia appears to be a guide as to the severity of the concussion. Headache is often the sole post-traumatic complaint, but shallow breathing, pallor, feeble pulse, reduced reflexes, and other signs of surgical shock may result. Visual impairments, equilibrium disturbances, and memory failure are common. The period of short-duration unconsciousness after concussion is attributed to the momentary compression of brain capillaries resulting in cerebral ischaemia/anaemia. Unconsciousness may be prolonged and reflex changes and even convulsions may manifest (especially in children). Any prolonged period of unconsciousness or inequality in pupil size indicates the need for neurologic consultation. Concussions can be graded on a scale of I–V (Table 6.1).

Table 6.1 Grading of concussion

- **Grade I**: Concussion in which a person is confused temporarily but does not display any memory changes
- **Grade II**: Concussion, brief disorientation and anterograde amnesia of less than 5 minutes duration are present
- **Grade III**: Concussion, retrograde amnesia and loss of consciousness for less than 5 minutes are present, in addition to the 2 criteria for a grade II concussion
- **Grade IV**: Similar to grade III with duration of loss of consciousness 5–10 minutes
- **Grade V**: Similar to grade III with duration of loss of consciousness longer than 10 minutes

Investigations

A CT scan of the brain or MRI is essential in these patients to determine if any bleeding over the brain surface or in the brain has occurred. The spinal fluid is always clear, and intracranial pressure is rarely elevated.

Treatment

All patients who sustain a concussion should be evaluated by a physician. After appropriate assessment, most persons with a mild concussion, who remain neurologically normal can generally be released from the emergency room but should be observed with a reliable companion for at least 12 hours. They should abstain from alcohol for several days and should be given mild analgesics for pain relief. However, patients with more severe symptoms after a concussion warrant hospital admission and observation. Such patients include those who remain drowsy or confused, have nausea or vomiting, severe headache, convulsions, leak of CSF from the ear or nose, and develop weakness or loss of feeling in the extremities, pupillary asymmetry, double vision, or other neurological symptoms. In these individuals, a more thorough evaluation and closer observation is indicated because the concussive event may have caused a skull fracture and/or a haemorrhage over the surface of the brain (epidural or subdural haematoma) or in the brain (cerebral contusion).

POST-CONCUSSIVE SYNDROME

Most patients have a favourable prognosis after mild brain injury; however, in some patients, a persistent and troublesome post-concussive syndrome develops, which causes a variety of subacute symptoms in addition to the acute symptoms. While no universally accepted definition exists, most of the literature defines the syndrome as the continuation of at least 3 of the following symptoms: headache, dizziness, fatigue, irritability, impaired memory and concentration, insomnia, and lowered tolerance for noise and light.

Clinical Evaluation

This complex constellation of pain-related and constitutional symptoms results from a perplexing combination of brain, extracranial, head, and cervical pathologic conditions (Table 6.2).

Table 6.2 Symptoms encountered in concussion and post-concussion syndrome

Acute	Dizziness or vertigo Headache Impairments of conscious awareness (disorientation, confusion, paralytic coma) Incoherent or slurred speech Incoordination and slowed motor responses Memory loss (retrograde and anterograde, impairment of 'working' memory) Nausea and vomiting (especially in children)
Subacute cranial trauma	Auditory impairment Benign positional vertigo Dizziness and light-headedness Headache Olfactory and gustatory deficits Tinnitus Visual complaints
Brain trauma	Affective disorders, posttraumatic stress disorder, increased anxiety Balance and coordination impairments Behavioural and personality changes, impairment or inability to concentrate and control mental functions (e.g., reverse order of set of digits) Seizures (rare) Sensory disturbances Sleep disturbance Tremor
Associated cervical trauma	Cervicogenic dizziness or vertigo Cervicogenic headache Neck pain Occipital neuralgia Reduced cervical spine range of motion
Nonspecific symptoms	Fatigue and loss of energy Sexual dysfunction

Evaluation can be problematic because objective documentable intracranial or extracranial findings are limited and interactions among a vast array of vague cognitive, behavioural, and emotional factors can produce subjective symptoms. Assessment and treatment may be further complicated by medicolegal considerations, since many patients are involved in litigation to recover compensation for their injuries. The factors that precipitate complicated and disabling long-term consequences and differentiate patients with a favourable prognosis from those who go on to have protracted post-concussive syndrome are not well understood.

Short & Long-term Effects of Concussion

About 50% of patients sustaining a cerebral concussion will have a complete resolution of symptoms within a few days of injury, although amnesia for events immediately before and after the injury is usually permanent. For the remaining 50% of concussion patients, particularly those with more severe forms of concussion, persistent changes may last 3 to 6 months or even up to a year after injury; in a few patients, a complete recovery never occurs.

Treatment

There is no widely agreed upon treatment for the post-concussion syndrome. Consequently, management of these problems must be highly individualized and is best done by a neurologist or neurosurgeon. Fortunately, most of the complaints associated with post-concussion syndrome resolve spontaneously without any specific therapy. Discrepancies between organic evidence and symptom presentation bring symptom origination into question. Manifestations most commonly occur during the initial weeks and resolve within 3 months after injury, yet symptoms persist in approximately one-third of patients with a mild head injury, often with objective cognitive deficits that may make assessment and treatment difficult.

DIFFUSE AXONAL INJURY

This has become recognized as one of the most important forms of primary injury to the brain and carries a greater risk of damage to the brain than intracranial haematomas. It is the commonest underlying pathology when there is no skull fracture and the commonest cause of vegetative state in patients surviving more than four weeks.

Mechanisms

Diffuse axonal injury can be caused by acceleration/deceleration injury or it can occur as a result of ischaemia. In acceleration injury the head is put into motion from a standstill position, as a result of which the different layers of the brain travel at different velocities with shearing effects and rotation of the brain within the skull. Strains of tentorium and falx during high-speed acceleration/deceleration produced in lateral motions of the head may cause the injuries. The shearing stresses between different layers of the brain result in petechial haemorrhages as well as diffuse axonal injury involving the white matter and brain stem. Diffuse axonal injury is characterized by extensive generalized damage to the white matter of the brain. This is seen in motor vehicle accidents where the car is hit from the back. In deceleration injury, the head is brought to a standstill from a moving position as in falls.

Neuropathologic Findings

Neuropathologic findings in patients with diffuse axonal injury were graded by Gennarelli and colleagues (Box).

- **Grade I**: Axonal injury mainly in parasagittal white matter of the cerebral hemispheres
- **Grade 2**: As in grade 1, plus lesions in the corpus callosum
- **Grade 3**: As in grade 2, plus a focal lesion in the cerebral peduncle

Clinical Features

Patients usually present with various states of altered sensorium since the time of injury. In the most extreme form, patients present with immediate prolonged unconsciousness from the moment of injury and subsequently remain vegetative or severely impaired. A marked discrepancy exists between the highly abnormal neurologic examination findings and the lack of findings on CT scanning.

CT scan (Fig. 6.2)

Of patients proven eventually to have diffuse axonal injury, 50–80% demonstrate a normal CT scan upon presentation. Delayed CT scan may be helpful in demonstrating oedema or atrophy, which are late findings. Characteristic CT findings in the acute setting are small petechial haemorrhages that are located at the gray-white matter junction, within the corpus callosum, and in the brainstem. Specific CT scan criteria for diffuse axonal injury are shown in Box and Marshall graded diffuse axonal injuries on the basis of CT scan findings Table 6.3.

CT scan criteria for diffuse axonal injury

- Single or multiple small intraparenchymal haemorrhages less than 2 cm in diameter in the cerebral hemisphere
- Intraventricular haemorrhage
- Haemorrhage within the corpus callosum
- Small focal areas of haemorrhage (< 2 cm in diameter) adjacent to the third ventricle
- Brainstem haemorrhage

Table 6.3 Marshall grading of diffuse brain injuries

Type I	Evidence of any significant brain injury is lacking
Type II	Either no midline shift or a shift less than 5 mm CSF cisterns at the base of the brain are widely patent No high-density or mixed-density lesions (contusions) of greater than 25 mL in volume
Type III	A midline shift of less than 5 mm with partial compression or absence of the basal cisterns No high- or mixed-density lesions with a volume greater than 25 mL
Type IV	Midline shift greater than 5 mm with compression or absence of the basal cisterns No lesions of high or mixed density greater than 25 mL

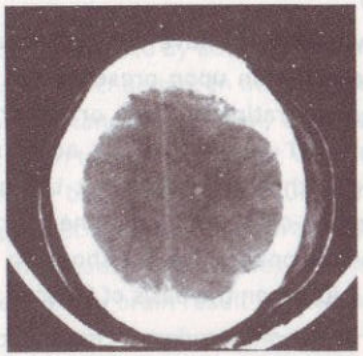


Fig. 6.2 Diffuse axonal injury

MRI

MRI is the investigation of choice for detection of diffuse axoal injury. The most common MRI finding is multifocal areas of abnormal signal (bright on T2- weighted images) at the white matter in the temporal or parietal corticomedullary junction or in the splenium of the corpus callosum. Other areas that frequently are abnormal include the dorsolateral rostral midbrain and the corona radiata. Recommended sequences include T1-weighted, proton density, T2-weighted, T2 gradient-echo, and diffusion-weighted images. T1-weighted images are helpful for anatomic localization; however, nonhaemorrhagic lesions may be iso-intense to surrounding tissue.

Haemorrhagic lesions appear hyper-intense on T1-weighted images. Nonhaemorrhagic lesions appear hyper-intense on T2-weighted sequences. Diffusion-weighted sequences can reveal hyperintensities in areas of axonal injury. Gradient-echo sequences are particularly useful in demonstrating the paramagnetic effects of petechial haemorrhages. Gradient-echo imaging often can demonstrate signal abnormality in areas that appear normal in T1 and T2-weigted spin-echo sequences. For this reason, it has become a mainstay of MR imaging of patients with suggested shearing-type injuries. The abnormal signal on gradient-echo images can persist for many years after the injury.

Outcome

The extent of the diffuse injury and the axonal damage determines the outcome. The more severe the injury is, more brain damage occurs with more axonal injury. This would be associated with higher morbidity and mortality and often patients remain in a vegetative state for long periods. Prognosis for full recovery often is poor.

CHAPTER 7

Intracranial Pressure

Increased ICP occurs in head injury either due to haematomas or cerebral oedema. Children are prone to develop significant oedema and this does not always occur as a result of severe head injury. Brain oedema could be localized around an area of brain damage or diffuse as seen in diffuse axonal brain injury.

PATHOPHYSIOLOGY

Normal ICP is < 10 mm Hg (136 mm water) and > 20 mm Hg is abnormal and a value of > 40 mm Hg is severe elevation. Monro-Kellie doctrine is the simple but vital concept for the understanding of dynamics of intracranial pressure. According to this principle, total volume of intracranial contents remains constant (blood volume + brain volume + CSF). Any addition in the cranial contents (i.e., haematoma) will lead to rise in ICP. However, it remains within normal limits until the patient reaches a critical point, then autoregulation and compensation mechanisms fail and patient decompensates. ICP value gets elevated at point of decompensation and the higher the ICP after head injury the worst will be the outcome. If untreated, this can give rise to brain herniation with its consequences (discussed below).

CLINICAL FEATURES

Most common clinical feature of raised ICP is headache that is worse on waking up in the morning and relieved by vomiting (intracranial pressure increases during sleep, some carbon dioxide retention? and vomiting induces hyperventilation and washes out the CO_2). Headache may be associated with nausea and vomiting that is also usually worse in the morning. Drowsiness is a late feature of raised ICP and should be considered carefully. Papilloedema is usally not seen in acute settings in head injury; however, it may develop in patients with chronic SDH. 6th nerve palsy, causing diplopia may occur in raised ICP due to stretching of the 6th nerve by caudal displacement of the brain stem. It is a false localizing sign. In infant, clinical features of raised ICP include excessive cry, failure to accept feed, irritability and bulging fontanelles.

HERNIATION SYNDROMES

The skull is a rigid bony compartment with only one exit (the foramen magnum). Severe brain swelling or a large intracranial haemorrhage (a space-occupying lesion) may cause displacement of the brain tissue downwards towards the foramen magnum. The inner, lower edge of a cerebral hemisphere becomes compressed against the sharp edges of the dural folds (causing uncal necrosis) or can be actually forced out under the free edge of the dura (causing uncal herniation). The cerebellum is similarly squeezed down into the foramen magnum (causing tonsillar coning and necrosis). Pressure on the brain stem and secondary brain stem haemorrhage may occur and are fatal complications of raised ICP. Several types of herniation syndrome can follow as a consequence of raised ICP

Transtentorial herniation

Transtentorial herniation occurs when the medial aspect of the temporal lobe (uncus) migrates across the free edge of the tentorium (tentorial hiatus). This causes pressure on the third cranial nerve, interrupting parasympathetic input to the eye and resulting in a dilated pupil. This unilateral dilated pupil is the classic sign of transtentorial herniation and usually (80%) occurs ipsilateral to the side of the transtentorial herniation. In addition to pressure on the third cranial nerve, transtentorial herniation compresses the brainstem (Box).

Subfalcine herniation

Subfalcine herniation occurs when the cingulate gyrus on the medial aspect of the frontal lobe is displaced across the midline under the free edge of the falx. This may compromise the blood flow through the anterior cerebral artery complexes, which are located on the medial side of each frontal lobe. Subfalcine herniation does not cause the same brainstem effects as those caused by transtentorial herniation.

Consequences of transtentorial herniation

- Compression of 3rd cranial nerve, initially causing ipsilateral dilatation of the pupil
- Pyramidal tract compression causes contralateral hemiparesis
- Lateral displacement of the brain stem may occur causing an ipsilateral hemiparesis
- Posterior cerebral artery kinking causing cerebral ischaemia and hemianopia may occur
- Brain stem compression will result in a deterioration of the level of consciousness leading to coma, hypertension and bradycardia (Cushing response and respiratory failure, which may be manifested as Cheyne-Stokes periodic breathing pattern).

Central herniation

Central herniation occurs when a diffuse increase in ICP occurs and each of the cerebral hemispheres is displaced through the tentorium, resulting in significant pressure on the upper brainstem.

Upward herniation

Upward, or reverse herniation occurs when either a large mass or increased pressure in the posterior fossa is present and the cerebellum is displaced in an upward direction through the tentorial opening. This also causes significant upper brainstem compression.

Tonsillar herniation

Tonsillar herniation occurs when increased pressure develops in the posterior fossa. In this form of herniation, the cerebellar tonsils are displaced in a downward direction through the foramen magnum, causing compression on the lower brainstem and upper cervical spinal cord as they pass through the foramen magnum. A slowly progressing tonsillar herniation may lead to abnormal neck posture and in a child it may cause neck tilt. Rapidly progressive lesion will cause respiratory failure, abrupt limb paresis and sensory disturbances.

Indications for ICP Monitoring

1. Abnormal scan, GCS < 8
2. Normal CT scan, GCS < 8 with any of the following two:
   - Age > 40 years
   - Posturing—Decorticate/Decerebrate
   - Hypotension
3. Inability to monitor GCS
   - Anesthesia for systemic injuries
   - Pharmacological paralysis for respiratory management
4. Need for treatment that might increase ICP
   - High levels of PEEP required for acute respiratory failure

ICP Monitoring Techniques

1. Intraventricular monitoring
   - Gold standard
   - CSF can be withdrawn as therapeutic measure for raised ICP
2. Subarachnoid
3. Epidural
4. Intraparenchymal

Stepwise Treatment of Established Raised ICP (see Chapter 10 also)

Begin treatment if ICP > 20–25 mm Hg

Goal is to maintain CPP > 70 mm Hg

Raised ICP
↓
CSF drainage through ventriculostomy
↓
Hyperventilation $paCO_2$ 30-35 mm Hg
↓
Mannitol 0.25–1.0 g/kg IV bolus
↓
High dose barbiturate therapy

Complications of ICP Monitoring

1. Infection
   - Duration > 5 days
   - Associated CSF leak

- Ventriculostomy
- Concurrent infection

2. Hemorrhage
3. Breakdown
4. Calibration

CHAPTER 8

Penetrating Head Injuries

Penetrating head trauma is a wound in which a projectile breaches the cranium but does not exit it and an injury in which the projectile passes entirely though the head, leaving both entrance and exit wounds, is described as ***perforating***. This distinction has prognostic implications as perforating wounds carry poor prognosis as compared to penetrating wounds. These wounds can be due to either high velocity injury (uncommon) or slow velocity injury as a result of penetration of the base of the scalp with sharp objects.

AETIOLOGY

Penetrating head injuries can be the result of numerous intentional or unintentional events, including missile wounds, stab wounds, and motor vehicle or occupational accidents (nails, screw-drivers). Stab wounds to the cranium typically are caused by a weapon with a small impact area and wielded at low velocity. The most common wound is a knife injury, although bizarre craniocerebral-perforating injuries have been reported that were caused by nails, metal poles, ice picks, keys, pencils, chopsticks, and power drills. The base of the skull is thin bone and could easily be penetrated especially in children with sharp objects as tree branches and knitting needles. These result in skull base fracture and damage to the brain overlying that area. Gunshot wounds to the head are also the most lethal of all firearm injuries with fatality rate of greater than 90% and two-thirds of the victims die before ever reaching a hospital.

PATHOPHYSIOLOGY

The pathological consequences of penetrating head wounds depend on the circumstances of the injury, including the properties of the weapon or missile, the energy of the impact, and the location and characteristics of the intracranial trajectory. Penetrations most commonly occur in the thin bones of the skull, especially in the orbital surfaces and the squamous portion of the temporal bone. The mechanisms of neuronal and vascular injury caused by cranial stab wounds may differ from those caused by other types of head trauma. Unlike missile injuries, no concentric zone of coagulative necrosis caused by dissipated energy and unlike in motor vehicle accidents, no diffuse shearing injury to the brain occurs. The local variations in thickness and strength of the skull and the angle of the impact determine the severity of the fracture and injury to the brain. Impacts striking the skull at nearly perpendicular angles may cause bone fragments to travel along the same trajectory as the penetrating object, to shatter the skull in an irregular pattern, or to produce linear fractures that radiate away from the entry defect. Grazing or tangential impacts produce complex single defects with both internal and external beveling of the skull, with varied degrees of brain damage.

Bullets can cause damage to brain parenchyma through three mechanisms (Box). The injury may range from a depressed fracture of the skull resulting in a focal haemorrhage to devastating diffuse damage to the brain.

Mechanism of injury (bullets)

- Laceration and crushing
- Cavitation
- Shock waves

Missile wounds. In missile wounds the amount of damage to the brain depends on multiple factors (Box).

Causative factors of injury (missiles)

- The kinetic energy imparted
- The trajectory of the missile & bone fragments through the brain
- Intracranial pressure (ICP) changes at the moment of impact
- Secondary mechanisms of injury

As a projectile passes through the head, tissue is destroyed and is either ejected out of the entrance or exit wounds or compressed into the walls of the missile tract. This creates both a permanent cavity that is 3–4 times larger than the missile diameter and a pulsating temporary cavity that expands outward. The temporary cavity can be as much as 30 times larger than the missile diameter and causes injury to structures a considerable distance from the actual missile tract (Box).

Mechanism of injury (missiles)

- The direct crush injury produced by the missile
- The cavitation produced by the centrifugal effects of the missile on the parenchyma
- The shock waves that cause a stretch injury

CLINICAL FEATURES

The clinical condition of the patient depends mainly on the mechanism (velocity, kinetic energy), anatomical location of the lesions, and associated injuries. Unless an associated haematoma or infarct is present, cerebral damage caused by stabbing is largely restricted to the wound tract.

A narrow elongated defect, or so-called slot fracture, sometimes is produced by a stab wound and is diagnostic when identified. However, in some cases in which skull penetration is proven, no radiological abnormality can be identified. Mortality is 17% in these cases and mostly related to vascular injury and massive intracerebral haematomas.

TREATMENT

Virtually all cranial gunshot victims are aggressively resuscitated upon initial arrival at the hospital. If a patients blood pressure and oxygenation can be maintained, an urgent CT scan of the brain is obtained. The decision to proceed with operative management of the gunshot wound is based on three factors:

- Level of consciousness (GCS)
- Degree of brainstem neurological function
- Findings on the CT scan

In virtually all patients who are deeply comatose with minimal evidence of brainstem function and no evidence of an intracranial haematoma that might be causing the coma, a fatal outcome is almost certain. In such patients, aggressive treatment is rarely pursued because of the futility of the situation. If, however, there is a haematoma seen on the CT scan, emergent craniotomy and clot evacuation is warranted, as some of these individuals will make a satisfactory recovery. For other patients who exhibit purposeful motor activity, urgent and aggressive neurosurgical care is provided.

OUTCOME

The predictors of poor neurological outcome or death after a gunshot wound to the head are shown in Box.

Predictors of outcome

- Initial low Glasgow coma score
- Older age
- Presence of low blood pressure
- Inadequate oxygenation after injury
- Dilated non-reactive pupils

Also, bullet trajectory through the brain has major significance. Bullets that traverse the brainstem, multiple lobes of the brain, or the ventricular system (chambers where cerebrospinal fluid is located) are particularly lethal. Many initial survivors will develop uncontrollable intracranial pressure with subsequent fatal outcome. Stab wounds to the temporal fossa are more likely to result in major neurological deficits because of the thinness of the temporal squama and the shorter distance to the deep brain stem and vascular structures. Patients in whom the penetrating object is left in place have a significantly lower mortality than those in whom the objects are inserted and then removed (26% versus 11% respectively).

CHAPTER 9

Complications of Head Injuries

CEREBRAL OEDEMA

Brain swelling (cerebral oedema) is a common and frequently fatal complication of head injury, which may develop within minutes or hours of injury (see Chapter 7). An oedematous brain shows visible enlargement of the surface convolutions (gyri) at the expense of obliteration of the intervening gaps (sulci) and compression of the fluid filled cavities (ventricles) deep within the brain. Swelling may accompany diffuse axonal injury or a space-occupying lesion such as an intracranial haematoma. In children, brain swelling may be the only identifiable feature of head injury. Focal oedema may be seen as localized poorly defined areas of low density. Diffuse swelling in children may be difficult to detect on CT scan. Emergency neurosurgical procedures frequently attempted include drainage of an ICH or removal of severely damaged brain tissue in an attempt to reduce intracranial pressure. Brain swelling is frequently the fatal complication even after such measures.

NECK INJURY

Neck is a very vulnerable area with easy access to vital structures such as the trachea, large vessels (carotid arteries, jugular [illegible]s). It can be involved in incisions, stabs, blows and manual pressure to the neck and can be very dangerous.

The cervical spine and spinal cord are also vulnerable (discussed in next section); at the same time, sensitive nervous connections and reflexes can be stimulated with fatal results (vagal reflex).

INFECTION

This can occur in compound skull fractures and skull base fractures leading to meningitis and brain abscess. Infections are particularly common after penetrating (open) head injury and after fractures which disrupt the nasal and frontal air sinuses. An adequate course of broad spectrum antibiotics according to culture and sensitivity is recommen-ded.

HYDROCEPHALUS

Hydrocephalus can be caused by blockage of the ventricular system by blood clot in cases of intraventricular haemorrhage or due to cicatrisation and fibrosis of subarachnoid space or the arachnoid villi along the sagittal sinus from deposition of blood products. Treatment options inculde ventriculoperitoneal shunts in obstructive hydrocephalus and theco-peritoneal shunt in communicating hydrocephalus.

CEREBROSPINAL FLUID LEAK

Cerebrospinal fistulae may occur in as many as 5–10% of patients as a result of fractures involving the skull base. Fractures involving ethmoid, frontal and sphenoid sinus will cause rhinorrhoea and fractures involving the temporal bone (crossing the internal ear and the middle ear with rupture of tympanic membrane) will cause otorrhoea. They may present either immediately or in a delayed fashion and in most of the cases these leaks are temporary. Approximately 80% of acute cases of CSF rhinorrhoea resolve spontaneously within 1 week and a 17% risk of meningitis exists with CSF rhinorrhoea. More than 95% of acute episodes of CSF otorrhoea resolve spontaneously within 1 week, and CSF otorrhoea is complicated by meningitis in fewer than 4% of cases. Prophylactic antibiotics have not been demonstrated to decrease this meningitis risk, although very few studies have examined this issue.

If the acute CSF leak does not resolve spontaneously, a lumbar subarachnoid drain may be placed for several days in an attempt to divert CSF and allow the fistula to close. If this fails, radiographic dye is introduced into the subarachnoid space via lumbar puncture (metrizamide cisternogram), and a high-resolution CT scan is performed in an attempt to identify the origin of the CSF fistula. MRI and CT cisternography are other options to investigate these patients to localize the site of leak.

Patients with persistent leak will require craniotomy repair of dural and bone defect. Delayed CSF fistulae may occur from 1 week after the initial injury to years later. These delayed fistulae are more difficult to treat and frequently require surgical intervention.

INFARCTION

It is rare complication and infarction in a typical vascular territory may suggest dissection of a vessel, such as the carotid artery after a direct blow to the neck. In these patients, carotid artery injury is to be ruled out.

POST-TRAUMATIC SEIZURES

Post-traumatic seizures (PTS) can be classified as immediate, early and late type. If the seizures occur within 24 hours of the injury, they are called immediate PTS. The PTS that occur within 1 week of the injury are termed early PTS, and seizures that occur more than 1 week after the injury are termed as late PTS.

Early post-traumatic seizures—Early post-traumatic seizures should be treated promptly as seizure activity is likely to further damage the already compromised brain. Use of phenytoin with maintenance of high therapeutic serum levels is recommended during the acute injury period. It is unclear if anticonvulsant therapy reduces the risk after one week post injury. Use of phenytoin during the first week after head injury reduces the risk of post-traumatic seizures by 70–75% during this period.

Late post-traumatic seizures—Healing and scarring of the meninges and brain surface may be the focus of late seizures. The use of anticonvulsants in patients with head injury is a controversial issue. No evidence exists that the use of anticonvulsants decreases the incidence of late-onset seizures in patients with either closed head injury or traumatic brain injury. The routine use of phenytoin in the first week following head injury decreases the incidence of early-onset (within 7 d of injury) seizures but does not change the incidence of late-onset seizures. In addition, the prevention of early post-traumatic seizures does not improve the outcome following traumatic brain injury. Therefore, the prophylactic use of anticonvulsants is not recommended for more than 7 days following traumatic brain injury and is considered optional in the first week following injury.

CHAPTER 10

Paediatric Head Injuries

Trauma is the commonest cause of death in childhood road traffic accidents followed by falls accounting for 80% of injuries and head injury is one of the most common reasons for children to attend emergency department. Children are more likely to have a minor injury and they are also more prone for non-accidental injury (child abuse). Assessment of children with head injureis is difficult and history is often vague and difficult to establish (i.e., whether or not there is a history of loss of consciousness). Furthermore, the adult Glasgow coma score is hard to apply to children, and there are various paediatric modifications. In children, threshold for investigations is low and the admission rate is comparatively high.

ANATOMICAL DIFFERENCES

The anatomical differences of the child brain render it more susceptible than the adult brain to certain types of injuries following head trauma. The head is larger in proportion to the body surface area, and stability is dependent on the ligamentous rather than bony structure. The paediatric brain has a higher water content, 88% versus 77% in adult, which makes the brain softer and more prone to acceleration-deceleration injury.

The water content is inversely related to the myelinization process. The unmyelinated brain is more susceptible to shear injuries. Infants and young children tolerate intracranial pressure (ICP) increases better because of open sutures.

ASSESSMENT

Even though most children have mild to moderate trauma, appropriate evaluation and therapy require considerable clinical judgment and are the most challenging problems confronting paediatricians and emergency physicians.

Assessment should follow same principles as adult and also it is important to know the weight of child to calculate fluid volumes and drug doses.

Airway and Breathing

Airway management in a child can be difficult due to:

- Large head relative to size of body
- Small oral cavity with large tongue
- Large angle of the jaw
- Larynx is cephalad
- Trachea is short

Remember Infants less than 6 months are obligate nose breathers. Uncuffed endotracheal tubes should be used in children before puberty

Circulation

Normal values for pulse and blood pressure vary with age (Table 10.1). Venous access in a child can be difficult, femoral or external jugular access may be required; if percutaneous cannulation fails, need to consider alternative route (Box) and initial resuscitation should be with a 20 mL/kg crystalloid bolus.

Table 10.1 Normal values for pulse and blood pressure in children

Age (in years)	Pulse rate	Systolic BP
Less than 1 year	120–140	70–90 mm Hg
2–5 years	100–120	80–90 mm Hg
5–12 years	80–100	90–110 mm Hg

Alternative routes for infusion

- Medial cephalic venous cut down
- Long saphenous venous cut down
- Intraosseous infusion

History Taking, Physical Examination & Neurologic Assessment

These procedures are invaluable to:

1. Determine the severity of the intracranial injury and identify those at risk for secondary injury
2. Identify injuries to other regions that may contribute to illness and death

Events surrounding the injury, such as the mechanism of injury, the time and the loss or presence of consciousness, should be obtained. However, reports may be inconsistent and hence unreliable. Fortunately, the progression of symptoms provides invaluable information to assist the physician in clinical decision-making. A brief seizure at the time of injury may not be clinically significant and may not necessitate therapy. However, one or more prolonged seizures associated with cardiorespiratory compromise necessitates prompt treatment. Many children will vomit two to three times after even a minor head injury. However, protracted vomiting and retching associated with other symptoms or signs indicate a more severe head injury and needs further investigation. Amnesia, irritability, lethargy, pallor or agitation may also indicate severe injury. The child medical history must be obtained; evidence of such conditions as a predisposition to seizures or a bleeding diathesis is important and would further dictate clinical management. The Glasgow coma scale is an important tool that influences treatment decisions and outcome and its modification may be used for children (Table 10.2).

The rest of the neurologic examination helps to determine the presence of focal neurologic signs that may signify an intracranial mass lesion or impending cerebral herniation from increased pressure. In all cases the physician should palpate scalp haematomas and contusions for underlying depressions, which signify a depressed skull fracture, and explore all full-thickness skull lacerations to ensure that the underlying bone is intact before suturing. Injuries to other areas such as the thorax or the abdomen should be sought and treated promptly since they may contribute to illness and death (Table 10.3). While examining the paediatric patients remember that they may be victims of child abuse and points to suspect this are shown in Table 10.4.

Table 10.2 Glasgow coma scale for children

	Score	> 1 year	0–1 year
Eye opening	4	Spontaneously	Spontaneously
	3	To verbal command	To shout
	2	To pain	To pain
	1	No response	No response
Best motor response	6	Obeys command	Obeys command
	5	Localizes pain	Localizes pain
	4	Flexion withdrawal	Flexion withdrawal
	3	Flexion abnormal (decorticate)	Flexion abnormal (decorticate)
	2	Extension (decorticate)	Extension (decorticate)
	1	No response	No response
Best verbal response	5	Appropriate words	Cries appropriately
	4	Inappropriate words	Cries inappropriately
	3	Screams	Crying/screaming
	2	Grunts	Grunts
	1	No response	No response

Table 10.3 Associated injuries

- Pulmonary contusion
- Pulmonary laceration
- Intrapulmonary haemorrhage
- Tracheobronchial tear
- Myocardial contusion
- Diaphragmatic rupture
- Great vessel disruption
- Oesophageal tears
- Liver tear
- Splenic rupture

Table 10.4 Diagnostic criteria for non-accidental injury

- Delay in seeking medical advice
- Vague or inconsistent account of the accident
- Discrepancy between the history and degree of injury
- Abnormal parental behaviour or lack of concern for the child
- Interaction between child and parents is abnormal
- Finger tip bruising over upper arm, trunk, face or neck. Bizarre injuries, bites, cigarette burns or rope marks
- Sharply demarked burns in unusual site
- Perioral injuries
- Retinal haemorrhages
- Rupture internal organs without a history of major trauma
- Perianal or genital injury
- Long bone fractures in children less than 3 years
- Injuries of differing ages

INVESTIGATIONS

Radiography plays no appreciable role in the evaluation and management of head trauma. CT scan is required for diagnosis and management of head injuries (details are discussed in Chapter 2). However, it serves as an adjunct to history-taking and physical and neurologic examination in the detection of skull fracture and thus enhances the care of the patient.

MANAGEMENT

Essential for successful management of children with head trauma are a proper clinical assessment of the primary injury and an appreciation of the potential for intracranial complications. Although most patients will not require hospital admission or diagnostic procedures, those at risk must be identified and observed closely. Fear of underlying brain injury and medicolegal reprisal, parental pressure and the difficulty in evaluating the patient may result in unnecessary investigations and prolonged periods of observation. Generalization of the experience with adults to children is inappropriate because of the differences in anatomy and physiologic response to cerebral trauma. Children may lose relatively large amounts of blood from scalp lacerations and subgaleal haematomas and present in haemorrhagic shock. Because a rapid pulse often signifies blood loss, other injuries should be sought.

The child with severe head injury should be kept in a normothermic state since increased cerebral metabolism may cause secondary brain injury.

Cerebral Oedema

Though children have a lower incidence of mass lesions than adults they are more likely to suffer from malignant cerebral oedema. Bradycardia with hypertension (a Cushing response) is usually a late response in children with increased intracranial pressure and therefore not very reliable. Early features of increased intracranial pressure include a decrease in the Glasgow coma score of 2 points or more, abnormality or changes in pupillary size and reaction to light, respiratory abnormalities and development of paresis in absence of shock, hypoxia or seizures. Since the risk of death increases with an intracranial pressure greater than 20 mm Hg, prompt, aggressive treatment in suspected cases is warranted (Table 10.5).

Table 10.5 Steps to control raised ICP

- Establish controlled hyperventilation (partial pressure of carbon dioxide 25 to 30 mm Hg)
- Elevate head of bed 30° to 45°, place head and neck in midline position
- Minimize stimuli (i.e., suctioning and movement)
- Restrict fluids to 60% of normal intake (except in cases of shock)
- Prescribe diuretics (mannitol, 0.5 to 1 g/kg intravenously, or furosemide, 1 to 2 mg/kg intravenously) in cases of documented deterioration despite above measures

Specific Considerations

Mild injury

Children with mild intracranial injury commonly do not pose a major therapeutic or diagnostic dilemma and can be discharged and observed at home. An instruction sheet should be given to the parents or caregiver concerning observation and precautions: Bring child immediately to emergency department if any of the signs and symptoms (Box) appear within the first 72 hours after discharge.

Signs and symptoms

- Vomiting
- Change in sensorium
- Severe headahce
- Any focal weakness
- Seizures
- Visual disturbance

Moderate injury

Close observation for at least 6 hours after injury is warranted. If the condition progressively improves during that time the child may be discharged home. A reliable caregiver should be in charge at home and be given an instruction sheet for observation and precautions. If any of these criteria are not met, the observation period in hospital should be extended to 24 hours. CT scanning and neurosurgical consultation may be necessary if the child's condition does not improve or deteriorates.

Severe injury

Severe head injuries in children are uncommon and often associated with motor vehicle accidents. Children with severe injury should be admitted to a tertiary care facility and be seen by a neurosurgeon. However, cardiorespiratory stabilization and treatment of increased intracranial pressure are more important than referral and should be started immediately.

OUTCOME

A child's brain has the ability to recover better from trauma, whether it is surgical or accidental, than adult brain. This is probably due to plasticity of the child brain. Also, children rehabilitate better and quicker than adults and hence outcome is better in children with same degree of injury as in adults.

CHAPTER 11

Geriatric Head Injuries

Injuries sustained by persons 65 years of age or older lead to higher morbidity rates than found in younger patients. Older trauma patients account for 25% of all trauma fatalities and use 33% of the total trauma health care resources. It is important to consider likely probabilities of cause and effect in the injured geriatric person. For example, when presented with a confused person after a fall: did the fall cause the confusion due to a head injury, or did the patient have a stroke that caused the fall? The stress of trauma can bring to the forefront signs and symptoms of a disease process that had previously been silent or ignored. Recognition of underlying health problems in the older trauma victim helps guide the most appropriate care.

PATHOPHYSIOLOGY AND CLINICAL CONSIDERATIONS

Self fall is the most common mechanism of injury in the elderly and causes include generalized weakness, environmental hazards, and orthostatic hypotension. Loss of consciousness resulting in a fall can be caused by heart rhythm disturbances, myocardial ischaemia or infarction, stroke, seizure, anaemia, or blood loss. Mentation can be altered significantly by alcohol and by prescription medication, which can lead to falls or other significant injuries in the elderly. Acute findings after a fall may have caused the fall or may be due to the mechanical fall itself. Brain aging results in cerebral atrophy and increased vascular fragility. Chronic subdural haematoma should be considered in an elderly patient presenting with changes in mental status, headache, disturbances in ambulation, or nonfocal neurological findings.

Spontaneous intracranial bleeding, strokes, or other cerebrovascular event may be the cause of fall in the elderly. Motor vehicle accidents are the second most common mechanism of injury (28%) in persons 65 years and older followed by pedestrians hit by motor vehicles (10%). Factors contributing to auto-pedestrian injuries seen in the elderly include impaired visual acuity and decreased peripheral vision, progressive deafness, postural kyphosis, altered gait, and diminished strength. Violence is an increasing cause of injury in elders including gunshot wounds or stabbings (8%). Physical abuse of dependent older adults requires special consideration.

ASSESSMENT

Immediate assessment should be guided by standard trauma protocols and airway managment is the top priority. Diagnostic workup of all trauma patients should include questions about the circumstances surrounding the injuries, the height of a fall, the speed of the vehicle, use of restraints or seat belts, associated loss of consciousness, and agility before and after the event. The diagnostic workup can be targeted from the history combined with the physical findings. Vital signs monitoring includes blood pressure, cardiac telemetry, and oxygen saturation. Mechanism of injury and a physical examination help determine the need for further diagnostic studies. Blood tests and urinalysis are indicated in patients with signs of anaemia, dehydration, or infection. Chest, spine, and extremity radiographs to be taken to rule out any injuries. Other imaging including computed tomography (CT), magnetic resonance imaging, ultrasonography, and vascular studies, should be considered when indicated.

Remember Age over 60 years is one of clinical indicators for ordering a head CT scan in the patient with minor head injury

MANAGEMENT

Aggressive intervention is warranted unless the patient is known to have a preexisting terminal disease or severe injuries with a low probability of survival. Treatment of the geriatric multiple trauma victim should follow standard trauma guidelines, including primary survey to assess critical injuries requiring immediate intervention, stabilization, and secondary survey for more complete patient assessment.

While managing elderly patients one should remember the fragility and limited physiological reserve of the geriatric patient.

Airway and Breathing

Establishing a patent airway is the first concern. Elderly patients may have upper airway obstruction due to relaxed musculature of the oropharynx and may have displaced dentures impairing airflow.

Circulation

Once adequate breathing and ventilation are secured, a search for blood loss, even in the face of normal vital signs as commonly used parameters to assess haemodynamic status can be misleading. Central venous pressure and arterial lines may be necessary to monitor fluid resuscitation. Fluids must be used with caution in the elderly patient, especially with underlying cardiovascular disease. Judicious use of intravenous crystalloid fluid is recommended, preferably Ringer lactate in the older patient, to avoid hyperchloraemia in the older patient with impaired renal capacity. For treatment of hypotension related to blood loss, infuse fluids at a rate of 2 L of crystalloid per unit of blood (packed red blood cells). Elderly patients may need platelets and fresh-frozen plasma earlier in the resuscitation than younger patients, especially in the presence of underlying liver dysfunction or coagulopathy.

Secondary Evaluation

After stabilizing the airway, breathing, and circulation (the ABCs), secondary evaluation and management should be directed by mechanism of injury and preexisting conditions. Complications during hospitalization and convalescence include infection, pulmonary embolus, and respiratory failure.

PROGNOSIS

Traumatic injuries result in a higher incidence of mortality in older patients; however, 57% to 67% of discharged patients 65 years and older live independently after discharge. Recognizing and treating the injuries aggressively increases the chance of survival to discharge. Rehabilitation improves mobility and independence.

FOLLOW-UP

Most homes are hostile to the elderly, especially the injured elderly and discharge planning should include an environmental assessment and recommend assistive technologies for home modifications. Home care services should provide nourishment, encourage physical activity, and prevent isolation.

CHAPTER 12

Rehabilitation in Head Injuries

The incidence of traumatic brain injuries is highest in people in the prime of their lives, coinciding with important events such as completing their education, developing their careers and establishing their families, and thus at a time when they are more likely to have financial problems. It has the potential to cause life-long impairments in physical, cognitive, behavioural and social function. The cognitive, behavioural and personality deficits are usually more disabling than the residual physical deficits.

COST FACTOR

Much of the disability (and costs) associated with these injuries is hidden, as survivors may have no physical evidence of their injury. However, the consequences can severely and permanently change a person's life, resulting in family disruption, loss of income and earning potential, and considerable expense over a lifetime.

EXTENT OF DISABILITIES

The range of severity varies from concussion through to persistent vegetative states with an extremely varied spectrum of possible lesions and resulting potential disabilities. Moreover, each person has a different set of premorbid abilities and a different psychosocial situation. Because of this, the goals of rehabilitation need to be holistic, long term and individualized to each survivor and his or her family. Though the family takes much of this responsibility, but some degree of support from medical and rehabilitation services will often be required for the rest of the person's life.

LONG-TERM PLANNING

As there is a long timeframe for improvement (at least two years), continuity of care is one of the most important goals in managing a person with traumatic brain injury. Explanations put forward for this continuous improvement are mostly speculative, incorporating concepts of various neural repair mechanisms, neuroplasticity and compensatory strategies. Longer-term improvement is thought to be the result of new learning. Rehabilitation is effective using an interdisciplinary approach, and close liaison with the patient, family and caregivers. The focus is on issues such as retraining in activities of daily living, pain management, cognitive and behavioural therapies, and pharmacological management.

RECOVERY AFTER BRAIN INJURY

Even though relatively much is known about different types of head injuries, it remains difficult to accurately predict in many patients the ultimate degree of neurological recovery. Despite very severe initial injuries, some patients make dramatic recoveries within several months to a year after injury.

PREDICTORS OF OUTCOME

Most patients with a mild head injury make a good recovery spontaneously and after severe head injury 33% make good recovery with treatment, 33% are left with varying degrees of disability, 33% die (in many cases this could be prevented).

Predictors of outcome

- Clinical parameters
- Older age (especially over approximately 50 to 60 years)
- Low Glasgow coma scale score (a deeper level of coma)
- Pupil dilatation
- Low blood pressure
- Inadequate oxygenation early after the injury
- Prolonged and difficult to control intracranial pressure

The strongest predictors of poor neurological and psychological outcome after head injury are mentioned in Box and importantly, these variables have an additive effect on morbidity and mortality; when multiple factors are present, such as older age, low GCS, pupil dilation & midline shift on CT scan, the chances for a good recovery are markedly diminished.

CT scan predictors

- Acute subdural haematoma
- Intracerebral haematomas
- Multiple contusions
- Subarachnoid haemorrhage
- Presence of compressed basilar cisterns
- Large degrees of brain shift from one side to the other (midline shift)

MEASURING SEVERITY OF TRAUMATIC BRAIN INJURY

Both in the acute stage and later rehabilitation, management is individualized to the person particular pattern of deficits or disabilities.

Table 12.1 Relation between Glasgow coma scale and morbidity and mortality

Glasgow coma scale	Good recovery or moderate disability (%)	Vegetative or dead (%)
11–15	91%	6%
8–10	59%	27%
5–7	28%	54%
3–4	13%	80%

Table 12.2 Functional score and outcome

Outcome	Score	Functional status
Good recovery	5	Normal or near normal
Moderate recovery	4	Disabled but independent
Severe disability	3	Dependent with physical or psychological disabilites or both
Persistent vegetative state	2	Dependent
Dead	1	

However, some broad outcomes to guide rehabilitation planning can be inferred from relatively simple injury severity markers (Table 12.1 & 12.2). The Glasgow coma scale is a quick and easy tool used to assess the severity of traumatic brain injury in the acute setting and gives a prognosis for survival rather than for functional outcomes.

Post-traumatic amnesia (PTA)

The duration of PTA is the best indicator of the extent of cognitive and functional deficits after TBI. PTA is defined as that period of time in which the brain is unable to lay down continuous day-to-day memory. The duration of PTA can be used as a guide to outcome, and correlates well with the extent of diffuse axonal injury and with functional outcomes. For example, one study found that 80% of patients with a PTA duration of less than 2 weeks had a good recovery, compared with 46% for those with a PTA duration between 4 and 6 weeks.

LONG-TERM CONSEQUENCES

Despite intensive intervention, long-term disability including significant neuropsychological problems and physical disabilities occurs in a large portion of the survivors of severe head injury (Table 12.3).

Minimally Responsive versus Persistent Vegetative State

Differentiating patients with a minimally responsive state from those with persistent vegetative states can be controversial for both clinical and legal reasons. Clinically, determining the cognitive capacity of a person with extremely severe motor deficits is a complicated issue requiring extended assessment. Persistent vegetative state indicates that the person, although showing signs of basic arousal, has been otherwise completely unable to interact with his or her environment for an extended period of time.

True permanent vegetative states are now exceedingly rare, due to a reduction in incidence of the condition and improved methods of assessment, and most patients become at least minimally responsive over time. This return of some level of consciousness has major implications, particularly as many of these people are young and are managed in facilities with limited rehabilitation opportunities or in high-care residential aged-care facilities.

Table 12.3 Long term consequences of head injury

Neurological impairment (motor, sensory & autonomic)	Headache and facial pain Motor function: impairment of coordination, balance, walking, hand function, speech Sensory loss: taste, touch, hearing, vision, smell Sleep disturbance, insomnia, fatigue
Medical complications	Spasticity, post-traumatic epilepsy, hydrocephalus, heterotopic ossification Sexual dysfunction
Cognitive impairment	Memory impairment Difficulty with new learning, attention and concentrations; reduced speed and flexibility of thought processing; impaired problem-solving skills Problems in planning, organizing, and making decisions Language problems: dysphasia, problem finding words, and impaired reading and writing skills Impaired judgment and safety awareness
Personality & behavioural changes	Psychiatric disorders: Anxiety, depression, post-traumatic stress disorder, psychosis, apathy, amotivational states Impaired social & coping skills, reduced self-esteem Altered emotional control; poor frustration tolerance & anger management; denial, and self-centeredness Reduced insight, disinhibition, impulsivity
Social/ economic problems	Unemployment and financial hardship, inadequate academic achievement Lack of transportation alternatives Inadequate recreational opportunities Difficulties in maintaining interpersonal relationships, marital breakdown Loss of independence Alcohol/drug abuse Crime

REHABILITATION

TBI rehabilitation often consists of two phases—inpatient and community management. Patients with significant neurocognitive impairment are best managed at a comprehensive rehabilitation unit for several weeks or months after they leave the hospital. Recovery of function from the time of discharge to 6 months post injury can be dramatic, even in some deeply comatose individuals. Improvement generally begins to plateau at 6 months post injury and is typically maximal by one year to 18 months. Rehabilitation is a team approach and needs participation and coordination between multiple specialities (Box).

The rehabilitation team

- Neurosurgeon
- Orthopaedic surgeon
- Patient and patient's family
- General practitioner
- Rehabilitation medicine physician
- Rehabilitation nurse
- Physiotherapist
- Occupational therapist
- Speech pathologist
- Social worker
- Neuropsychologist
- Clinical psychologist
- Vocational rehabilitation services and counsellors

Inpatient Management

Inpatient management is required for those with more severe and acute physical, cognitive and/or behavioural deficits. The focus is on issues such as PTA monitoring, retraining in activities of daily living, pain management, cognitive and behavioural therapies, pharmacological management, assistive technology (e.g., prescription wheelchairs and gait aids), environmental manipulation (e.g., installation of lifts, ramps and rails, and bathroom alterations), as well as family education and counselling. Patients may also require retraining in daily living activities for home and community living (e.g., household tasks such as doing the laundry, and community living skills such as crossing roads, banking, etc.).

Community Rehabilitation

Community rehabilitation follows discharge from an inpatient rehabilitation service. Helping a person with TBI return to maximum independence and participation in the community is an extremely difficult task. Family support, education and counselling are vital and likely to be needed for a prolonged period. The quality and availability of community services can be less than ideal and issues of cost may limit access. This applies particularly to adapted lifestyle-sustaining services.

Rehabilitation of Mild Traumatic Brain Injury

It is estimated that 70%–75% of all traumatic brain injuries fall into the mild category. While they rarely require inpatient rehabilitation, patients commonly report cognitive and behavioural changes from which they recover within 3 months. Out of these, 10%–15% will remain symptom-atic in the longer term with a persisting post-concussion syndrome (complaints including headache, cervical pain, vestibular symptoms; changes in taste and hearing; difficulty with attention and memory; and irritability, insomnia and sleeping difficulties).

Interpersonal relationships and work may also be affected. Treatment of these patients involves patient and family education, reassurance and psychological support.

Rehabilitation of Moderate and Severe Traumatic Brain Injury

Patients within these categories show a broad range of possible outcomes, and it is generally not possible to predict the extent of recovery in the initial weeks after the trauma. Many patients with a dire early prognosis successfully return to competitive employment. Most will be independently mobile and be physically, if not cognitively, capable of self-care and normal community living.

Determining the combination of cognitive, behavioural and physical deficits is an important first step in setting goals for rehabilitation. Prioritizing goals should be undertaken with the assistance of both the person and their family. Patients for whom there is no support, or for whom such support is inadequate or inappropriate, fare worse despite the degree or type of rehabilitation.

Retraining and Reskilling

Return to work is an important factor that contributes to satisfaction and quality of life. Vocational and leisure options may include retraining, reskilling, on-the-job training or supported employment services.

Behavioural Management

Behavioural changes may alienate family and friends, with families sometimes perceiving the person as a difficult stranger. Aggression, substance misuse or lack of empathy particularly strain relationships for others, who may see the patient as unmotivated and lazy. Behavioural management may be necessary to increase independence and reduce maladaptive social behaviour: agitation, irritability, combative outbursts, lethargy and abnormal or foul language.

Drug Therapy

Drugs are sometimes useful in the management of mood disorders, such as depression and anxiety following trauma. The newer antidepressants, such as the selective serotonin reuptake inhibitors (SSRIs), are most commonly used. Mood stabilizers (e.g., carbamazepine and sodium valproate) can be used to reduce the anger dyscontrol sometimes exhibited by those with executive dysfunction. When anticonvulsant medication is required, phenytoin is not commonly used owing to its adverse cognitive effects, but carbamazepine and sodium valproate can be used.

Rehabilitation of Children with Traumatic Brain Injury

Children have better outcomes than adults after brain injury. While fewer focal deficits may be apparent, higher cognitive deficits may not become apparent until later in the child development. These children may have impaired new learning, inability to take on social cues, and behavioural, educational and schooling problems. These problems pose many challenges for parents, teachers and healthcare workers particularly in coping with changed academic aspirations for children.

CHAPTER 13

Prevention of Head Injuries

In the United States, Canada and elsewhere, public education initiatives on brain and spinal cord injury such as the Think First program, and enactment of safety legislation have impacted on the problem of CNS trauma. Unfortunately, in India no such programmes are available. From 1982 to 1992, seat belt use among drivers and passengers in the US increased from 11% to 66%.

In 1992, air bags were estimated to have saved over 550 lives and prevented 40,000 serious injuries. Fatalities from motorcycle use have fallen from over 4600 in 1982 to 2400 in 1992 in conjunction with implementation of motorcycle helmet laws. Increasing use of infant restraint seats from 1982 to 1992 was estimated to have saved over 2000 infant lives.

The pervasive problem of alcohol-related traffic accidents is also starting to diminish due to broad educational efforts and strict enforcement of drunk driving laws. Between 1982 and 1992, alcohol-related traffic deaths fell by over 30% despite a rising number of vehicles on the road.

PREVENTION

Passenger seat belts and airbags are useful in preventing head injury, also, helmet use by children and adolescents during certain sport activities will reduce risk of head injuries.

Education regarding avoidance of alcohol and drug use will also help in decreasing the incidence of alcohol and drug-related accidents. Children younger than 12 years should ride in the back seat of the car away from the airbag.

HELMETS: MYTHS AND FACTS

During a road traffic accident the possibility of impact on head is about 40%, therefore it is very important to prevent head injury. Although many risk factors for crashes have been identified, such as alcohol use, driver fault, speeding, vehicle design, there has been little reported success in reducing two wheeler crash rates.

The most successful injury prevention approach has been reduction of severity of injury after the crash and the countermeasure most employed worldwide for this purpose has been helmets.

The history of helmet dates back to 1935, when Col. TE Lawrence (Lawrence of Arabia) was fatally injured in a motorcycle accident, one of the several doctors attending him was a young neurosurgeon, Hugh Cairns. He was moved by the tragedy in a way that was to have far-reaching consequences. His research concluded that the adoption of crash helmets as standard by both military and civilian motorcyclists would result in considerable saving of life.

After implementation of the helmet use law worldwide in many nations motorcycle crash fatalities due to head injury decreased by as much as 40%.

Traumatic brain injury admissions, because of two wheeler crashes, reduces to one third if helmet is universally used. Riders wearing helmets suffer fewer skull fractures, fewer intracranial injuries, less frequent and shorter periods of loss of consciousness, more favourable coma scores and shorter hospital stays. Besides head injuries other injuries prevented by a proper helmet are facial and cervical spine injuries.

Despite proven benefits of helmet use, there is high level of reluctance among public due to widely prevalent myths regarding helmet use (Box).

Myths and facts about helmet use

- *Myth*: Helmet is not necessary at low speeds
- *Fact*: Even speeds as low as 17 kmph can result in serious head injury and death

- *Myth*: Helmet straps cause strangulation
- *Fact*: No such evidence has been found

- *Myth*: Helmets cause neck injury
- *Fact*: No scientific study to date has found any contribution to neck injury. In any case helmeted riders often survive to complain of neck pains, unhelmeted riders don't

- *Myth*: Helmets reduce range of vision
- *Fact*: Most helmets restrict peripheral vision by less than 5%, that is much less than spectacle frames and goggles. Standard helmets do not obstruct critical or even potential vision

- *Myth*: Helmets cut out warning sounds of other vehicles
- *Fact*: Any sound, loud enough to be heard over the noise of motorcycle and wind will be loud enought to be heard inside the helmet. In fact, helmet will give protection to both eyes and ears.

- *Myth*: Helmet use results in baldnes, headache, allergy and fatigue
- *Fact*: There is no difference in incidence of baldness, headache, allergy and fatigue in helmeted and non-helmeted riders

- *Myth*: Helmet is not necessary for pillions
- *Fact*: Pillions suffer more severe head injury than rider; hence, it is more necessary for them

- *Myth*: There is increase in temperature inside the helmet
- *Fact:* Temperature inside the helmet stabilizes slightly above body temperature. The insulation of the helmet makes its interiors more subject to body temperature than to outside temperature

If people are made aware of how disabling head injury can be, more and more people will voluntarily wear helmet. The simple deed of wearing a helmet can be life saving.

CHAPTER 14

Brain Death

DEFINITION

Brain death is defined as an irreversible and complete loss of function of the entire brain including the brain stem.

CLINICAL EXAMINATION

The clinical examination of patients who are presumed to be brain dead must be performed with precision. The declaration of brain death requires not only a series of careful neurologic tests but also the establishment of the cause of coma, the ascertainment of irreversibility, the resolution of any misleading clinical neurologic signs, the recognition of possible confounding factors, the interpretation of the findings on neuroimaging, and the performance of any confirmatory laboratory tests that are deemed necessary.

NEUROLOGIC EXAMINATION

Neurologic examination to determine whether a patient is brain dead can proceed only if the following prerequisites are met: the ruling out of complicated medical conditions that may confound the clinical assessment, particularly severe electrolyte, acid–base, or endocrine disturbances; the absence of severe hypothermia, defined as a core temperature of 32°C or lower; hypotension; and the absence of evidence of drug intoxication, poisoning, or neuromuscular blocking agents.

Complete clinical neurologic examination includes documentation of coma, the absence of brainstem reflexes, and apnoea. The examina-

tion of brainstem reflexes requires the measurement of reflex pathways in the mesencephalon, pons, and medulla oblongata. As brain death occurs, patients lose their reflexes in a rostral-to-caudal direction, and the medulla oblongata is the last part of the brain to cease to function. Several hours may be required for the destruction of the brain stem to be complete, and during that period, there may still be medullary function. An obligatory prerequisite for the diagnosis of brain death is the proof of severe primary or secondary brain damage.

Table 14.1 Clinical criteria for brain stem death in adults and children

Coma
Absence of motor response
Absence of pupillary response to light and pupils at mid-position with respect to dilatation (4–6 mm)
Absence of corneal reflex
Absence of corneal response
Absence of gag reflex
Absence of coughing in response to tracheal suctioning
Absence of sucking and rooting reflexes
Absence of respiratory drive at a $PaCO_2$ that is 60 mm Hg or 20 mm Hg above normal baseline values
Interval between two evaluations, according to patient's age
Term to 2 months old, 48 hr
> 2 months to 1 year old, 24 hr
>1 year to 18 years old, 12 hr
> 18 years old, interval optional
Confirmatory tests (Table 14.2)
Term to 2 months old, 2 confirmatory tests
> 2 months to 1 year old, 1 confirmatory test
>1 year to 18 years old, optional
> 18 years old, optional

Table 14.2 Confirmatory testing for a determination of brain death

▪ Cerebral angiography
▪ Electroencephalography
▪ Transcranial Doppler ultrasonography
▪ Cerebral scintigraphy (technetium Tc 99m hexametazime)

STEPS OF EXAMINATION FOR BRAIN DEATH

Step 1

The physician determines that there is no motor response and the eyes do not open when a painful stimulus is applied to the supraorbital nerve or nail bed.

Step 2

A clinical assessment of brainstem reflexes is undertaken.

The absence of grimacing or eye opening with deep pressure on both condyles at the level of the temporomandibular joint (afferent nerve V and efferent nerve VII).

The absent corneal reflex elicited by touching the edge of the cornea (V and VII).

The absent light reflex (II and III).

The absent oculovestibular response toward the side of the cold stimulus provided by ice water (pen marks at the level of the pupils can be used as reference; VIII and III and VI).

The absent cough reflex elicited through the introduction of a suction catheter deep in the trachea (IX and X).

Step 3

The apnoea test is performed; the disconnection of the ventilator and the use of apnoeic diffusion oxygenation require precautionary measures. The core temperature should be 36.5°C or higher, the systolic blood pressure should be 90 mm Hg or higher, and the fluid balance should be positive for six hours. After pre-oxygenation (the fraction of inspired oxygen should be 1.0 for 10 minutes), the ventilation rate should be decreased. The ventilator should be disconnected if the partial pressure of arterial oxygen reaches 200 mm Hg or higher and if the partial pressure of arterial carbon dioxide reaches 40 mm Hg or higher. The oxygen catheter should be at the carina (delivering oxygen at a rate of 6 liters per minute). The physician should observe the

chest and the abdominal wall for respiration for 8 to 10 minutes and should monitor the patient for changes in vital functions. If there is a partial pressure of arterial carbon dioxide of 60 mm Hg or higher or an increase of more than 20 mm Hg from the normal baseline value, apnoea is confirmed.

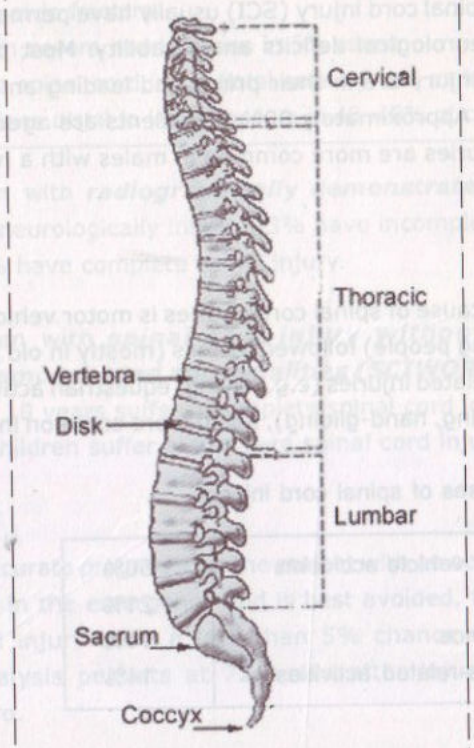
IMPLICATIONS

After the clinical criteria of brain death have been met, the physician should inform the next of kin, who can be approached about organ donation. The physician is required to abide by state law with respect to organ donation. Organ-procurement agencies must be notified to request the donation of organs. If the legal next of kin declines to donate organs, it is good medical judgment to discontinue mechanical ventilation. When mechanical ventilation and support are continued because of ethical or legal objections to their discontinuation, what usually follows is an invariant heart rate from a differentiated sinoatrial node, structural myocardial lesions leading to a marked reduction in the ejection fraction, decreased coronary perfusion, the need for increasing use of inotropic drugs to maintain blood pressure, and a fragile state that leads to cardiac arrest within days or weeks.

Further Reading

- H Richard Winn, *Youmans Neurological Surgery*, 5th edition, 2004.
- The Brain Trauma Foundation. The American Association of Neurological Surgeons. The Joint Section on Neurotrauma and Critical Care. Guidelines for management of severe head injuries. *J Neurotrauma*. 2000 Jun-Jul;17(6–7)

Section 2 : Spine injuries



CHAPTER 15

Epidemiology of Spine Injuries

Patients with spinal cord injury (SCI) usually have permanent and often devastating neurological deficits and disability. Most patients with a cervical spine injury are in their prime and leading an active lifestyle prior to injury. Approximately 80% of patients are aged 18–25 years. Spinal cord injuries are more common in males with a male-to-female ratio of 4:1.

CAUSES

Most common cause of spinal cord injuries is motor vehicular accidents (mostly in young people) followed by falls (mostly in old people) (Table 15.1), sports-related injuries (e.g., diving, equestrian activities, football, gymnastics, skiing, hand-gliding), again more common in young adults.

Table 15.1 Causes of spinal cord injuries

Motor vehicle accidents	50%
Falls	21%
Violence	14%
Sports-related activities	14%

Cervical spine is more commonly involved, followed by dorsolumbar junction (Table 15.2).

Table 15.2 Regions involved in spinal injuries

Cervical spine	40%
Thoracic spine	10%
Thoracolumbar junction	35%
Lumbar spine	3%

Facts and figures

- Multi-system trauma in 80% of cases
- Very often concurrent with long bone fractures
- Often concurrent with thoracic and abdominal visceral injuries, pelvis fracture
- Along with severe head injuries in 5% cases
- Complete spinal cord injury (total sensory and motor function loss distal to injury) occurs in 43–46% of cases

Among children with ***radiographically demonstrated abnormalities*** 50% are neurologically intact, 33% have incomplete spinal cord injury and 17% have complete spinal injury.

Among children with ***spinal cord injury without any radiographically demonstrated abnormalities (SCIWORA)*** 50–75% of children under 10 years suffer a complete spinal cord injury and 11% of teen-aged children suffer a complete spinal cord injury.

PROGNOSIS

Providing an accurate prognosis for the patient with an acute SCI usually is not possible in the emergency and is best avoided. Patients with a complete cord injury have a less than 5% chance of recovery. If complete paralysis persists at 72 hours after injury, recovery is essentially zero.

The prognosis is much better for the incomplete cord syndromes. If some sensory function is preserved, the chance that the patient will eventually be able to walk is greater than 50%. Ultimately, 90% of patients with SCI will return to their homes and will regain independence.

MORTALITY

In the early 1900s, the mortality rate 1 year after injury in patients with complete lesions approached 100%. Much of the improvement since then can be attributed to the introduction of antibiotics to treat pneumonia and urinary tract infection. Currently, the 5-year survival rate for patients with a traumatic quadriplegia exceeds 90% and hospital mortality rate for isolated acute SCI is low. The leading cause of death at present is pneumonia, followed by non-ischaemic heart disease and sepsis.

CHAPTER 16

Anatomy and Pathophysiology of Spinal Injuries

SPINAL CORD

The normal spinal cord is represented in the figure as a cross section (Figure 16.1). As the injury to different regions of the spinal cord will produce predictable patterns, a knowledge of spinal cord anatomy is important. The spinal cord is divided into 31 segments, each with a pair of anterior (motor) and dorsal (sensory) spinal nerve roots. On each side, the anterior and dorsal nerve roots combine to form the spinal nerve as it exits from the vertebral column through the neural foramina. The spinal cord extends from the base of the skull and terminates near the lower margin of the L1 vertebral body. Thereafter, the spinal canal contains the lumbar, sacral, and coccygeal spinal nerves that comprise the cauda equina.

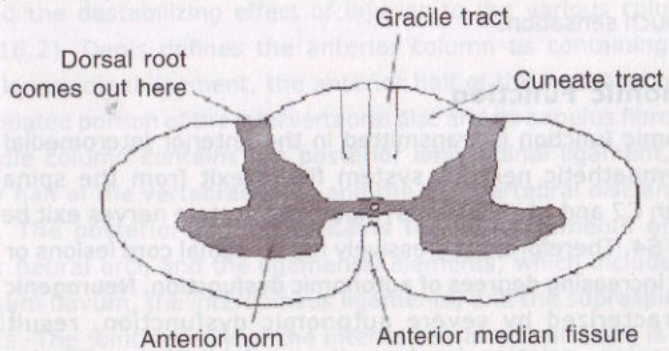


Fig. 16.1 Section of spinal cord

Spinal injuries proximal to L1, above the termination of the spinal cord, often involve a combination of spinal cord lesions and segmental root or spinal nerve injuries. The spinal cord itself is organized into a series of tracts or neuropathways that carry motor (descending) and sensory (ascending) information. These tracts are organized anatomically within the spinal cord. The corticospinal tracts are descending motor pathways located anteriorly within the spinal cord. Axons extend from the cerebral cortex in the brain as far as the corresponding segment, where they form synapses with motor neurons in the anterior (ventral) horn. They decussate (cross over) in the medulla prior to entering the spinal cord. The dorsal columns are ascending sensory tracts that transmit light touch, proprioception, and vibration information to the sensory cortex. They do not decussate until they reach the medulla. The lateral spinothalamic tracts transmit pain and temperature sensation. These tracts usually decussate within 3 segments of their origin as they ascend. The anterior spinothalamic tract transmits light touch. Autonomic function traverses within the anterior interomedial tract. Sympathetic nervous system fibers exit the spinal cord between C7 and L1, while parasympathetic system pathways exit between S2 and S4. Injury to the corticospinal tract or dorsal columns, respectively, results in ipsilateral paralysis or loss of sensation of light touch, proprioception, and vibration. Unlike injuries of the other tracts, injury to the lateral spinothalamic tract causes contralateral loss of pain and temperature sensation. Because the anterior spinothalamic tract also transmits light touch information, injury to the dorsal columns may result in complete loss of vibration sensation and proprioception but only partial loss of light touch sensation.

Autonomic Function

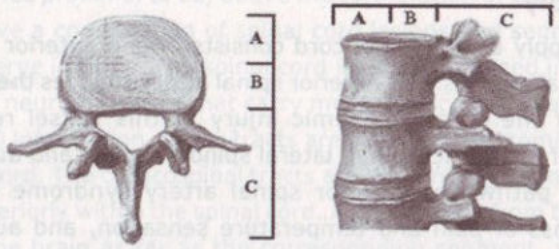
Autonomic function is transmitted in the anterior interomedial tract. The sympathetic nervous system fibers exit from the spinal cord between C7 and L1. The parasympathetic system nerves exit between S2 and S4. Therefore, progressively higher spinal cord lesions or injury causes increasing degrees of autonomic dysfunction. Neurogenic shock is characterized by severe autonomic dysfunction, resulting in hypotension, relative bradycardia, peripheral vasodilation, and hypothermia. It does not usually occur with SCI below the level of T6. Shock associated with an SCI involving the lower thoracic cord must be considered haemorrhagic until proven otherwise.

Blood Supply

The blood supply of the spinal cord consists of one anterior and two posterior spinal arteries. The anterior spinal artery supplies the anterior two thirds of the cord. Ischaemic injury to this vessel results in dysfunction of the corticospinal, lateral spinothalamic, and autonomic interomedial pathways. Anterior spinal artery syndrome involves paraplegia, loss of pain and temperature sensation, and autonomic dysfunction. The posterior spinal arteries primarily supply the dorsal columns. The anterior and posterior spinal arteries arise from the vertebral arteries in the neck and descend from the base of the skull. Various radicular arteries branch off the thoracic and abdominal aorta to provide collateral flow. The primary watershed area of the spinal cord is the midthoracic region. Because of its peculiar blood supply, vascular injury may cause a cord lesion at a level several segments higher than the level of spinal injury. For example, a lower cervical spine fracture may result in disruption of the vertebral artery that ascends through the affected vertebra resulting in ischaemic injury to high cervical cord. At any given level of the spinal cord, the central part is a watershed area. Cervical hyperextension injuries may cause ischaemic injury to the central part of the cord, causing a central cord syndrome.

Three-column Model of the Spine

In 1983, Denis proposed the 3-column model of the spine, which described both the functional units that contribute to the stability of the spine and the destabilizing effect of injuries to the various columns (Figure 16.2). Denis defines the anterior column as containing the anterior longitudinal ligament, the anterior half of the vertebral body, and the related portion of the intervertebral disc and its annulus fibrosus. The middle column contains the posterior longitudinal ligament, the posterior half of the vertebral body, and the intervertebral disc and its annulus. The posterior column contains the bony elements of the posterior neural arch and the ligamental elements, which include the ligamentum flavum, the interspinous ligaments, and the supraspinous ligaments. The joint capsule of the intervertebral articulations is also part of the posterior column. Disruption of two or more columns results in an unstable configuration.



A. Anterior column, B. Middle column, C. Posterior column

Fig. 16.2 Three-column model of spinal cord injuries

PATHOPHYSIOLOGY

Spinal cord injury can be primary or secondary (as for head injuries—Chapter 2) and another specific type of injury is SCIWORA (spinal cord injury without radiologic abnormality).

Primary Injuries

Primary SCIs arise from mechanical disruption, transection, or distraction of neural elements and usually occurs with fracture and/or dislocation of the spine. However, primary SCI may occur in the absence of spinal fracture or dislocation. Other causes of primary injuries are penetrating injuries due to bullets or weapons; displaced bony fragments can penetrate spinal cord and/or segmental spinal nerves. Spinal epidural haematomas can cause acute cord compression and injury. Longitudinal distraction with or without flexion and/or extension of the vertebral column may result in primary SCI without spinal fracture or dislocation. This initial mechanical trauma typically leads to direct compression of neural elements by bone fragments, disc material, and ligaments and damages both the central and peripheral nervous systems.

Secondary Injuries

Major causes of secondary SCIs are ischaemic injury to the spinal cord caused by arterial disruption, arterial thrombosis, or hypoperfusion due to shock. Loss of autoregulation and spinal shock will cause systemic hypotension and ischaemic spinal cord damage. Other causes include toxic metabolic compounds, and electrolyte changes leading to a secondary injury cascade. Hypoperfusion of gray matter may extend to the surrounding white matter and alter the propagation of action potentials along the axons, contributing to spinal shock. Glutamate is a key element in the excitotoxicity and neuronal damage. In all acute cord syndromes, the full extent of injury may not be apparent initially. Incomplete cord lesions may evolve into more complete lesions and the injury level rises 1 or 2 spinal levels during the hours to days after the initial event. A complex cascade of pathophysiologic events related to free radicals, vasogenic oedema, and altered blood flow accounts for this clinical deterioration. Normal oxygenation, perfusion, and acid-base balance are required to prevent worsening of the SCI.

SCIWORA

The term SCIWORA (spinal cord injury without radiologic abnormality) was first coined in 1982 by Pang and Wilberger. The spinal cord is tethered more securely than the vertebral column. Longitudinal distraction of the spinal cord with or without flexion and/or extension of the vertebral column may result in SCIWORA.

Spinal Shock (neurogenic shock)

Spinal shock is defined as the complete loss of all neurologic function, including reflexes and rectal tone, below a specific level associated with autonomic dysfunction. Neurogenic shock refers to the haemodynamic triad of hypotension, bradycardia, and peripheral vasodilation resulting from autonomic dysfunction and the interruption of sympathetic nervous system control in acute SCI.

CHAPTER 17

Evaluation of Spine Injuries

Management of spine injuries starts at the scene of accident. First and foremost step is to maintain in-line spinal immobilization in any trauma victim followed by support of neck with stiff collar and sandbags. Patient should be transported on spinal board and should be moved like a log of wood. The goals for the emergency physician are to establish the diagnosis and initiate treatment to prevent further neurological injury from either pathologic motion of the injured vertebrae or secondary injury from the deleterious effects of cardiovascular instability or respiratory insufficiency. All patients with head trauma or maxillofacial trauma should be presumed to have an unstable spine particularly cervical spine injury until positively excluded. Absence of neurological deficit does not exclude spine injury. Examination of the spine is impaired in comatose patients. Waiting for several hours to exclude spine injury and leaving patient in hard collar and on spinal board is poor management.

PRIMARY SURVEY

Principles of treatment of these are as for head injury patients (Chapter-3). Maintenance of the airway is to be done carefully in these patients (Chapter 3). Two main concerns with spinal injuries are hypotension and respiratory failure.

Blood Pressure Management

In all patients with SCI and hypotension, a diligent search for sources of haemorrhage must be made before hypotension is attributed to neurogenic shock as in acute SCI, shock may be neurogenic, haemorrhagic, or both. Disruption of autonomic pathways prevents tachycardia and peripheral vasoconstriction that normally characterizes haemorrhagic shock. The presence of vital sign confusion and a high incidence of associated injuries requires a diligent search for occult sources of haemorrhage. The following points will help in distinguishing haemorrhagic shock from neurogenic shock:

- Neurogenic shock occurs only in the presence of acute SCI above T6.
- Hypotension and/or shock with acute SCI at or below T6 is caused by haemorrhage.
- Hypotension with a spinal fracture alone, without any neurologic deficit or apparent SCI, is invariably due to haemorrhage.

Patients may be both hypotensive and bradycardic (NB: Not a feature of hypovolaemia due to visceral injuries, therefore suspect spinal cord injury).

The therapeutic goal for neurogenic shock is adequate perfusion with the following parameters:

- Systolic blood pressure (BP) should be 90–100 mm Hg.
- Heart rate should be 60–100 beats per minute in normal sinus rhythm.
- Haemodynamically significant bradycardia may be treated with atropine.
- Urine output should be more than 30 mL/h. Placement of a Foley catheter to monitor urine output is essential. Rarely, inotropic support with dopamine is required. It should be reserved for patients who have decreased urinary output despite adequate fluid resuscitation. Usually, low doses of dopamine in the 2- to 5-µg/kg/min range are sufficient.

Respiratory Management

The clinical assessment of pulmonary function in acute SCI begins with careful history taking regarding respiratory symptoms and a review of underlying cardiopulmonary comorbidity such as chronic obstructive pulmonary disease or heart failure. Carefully evaluate respiratory rate, chest wall expansion, abdominal wall movement, cough, and chest wall and/or pulmonary injuries. Arterial blood gas (ABG) analysis and pulse oximetry are especially useful because the bedside diagnosis of

Incomplete

There is some preservation of sensory/motor function below level of injury. Incomplete are of several types depending on the extent of injury (Box). These syndromes are discussed later in detail.

Types of incomplete injuries
▪ Anterior cord syndrome ▪ Central cord syndrome ▪ Brown-Sequard syndrome ▪ Posterior cord syndrome ▪ Conus medullaris syndrome

Examination

The purpose of secondary survey is to assess the extent and level of spine injury. Details of neurological examination are discussed in Chapter 2 and more relevant features are highlighted here (Table 17.4 to Table 17.9). In most clinical scenarios, the emergency physician should use a best-fit model to classify the SCI syndrome. At the end of evaluation patient should be classified functionally by classification system of Frankel in 5 grades (A–E) (Table 17.10).

Table 17.4 Level of injury and clinical manifestations

Vertebral level	Damage
Above C5	Complete quadriplegia, sometimes respiratory paralysis, often fatal
C5–C6	Legs and (usually) wrists and hands paralyzed, but shoulder movement and elbow flexion usually present
Thoracic (T1–T12)	Leg muscles paralyzed
Thoracolumbar (T12-L1) Lumbar (L2-L5)	Possible conus medullaris syndrome Possible cauda equina syndrome (hypo or areflexic pareiss of lower extremities)

Table 17.5 Muscle groups and nerve with root value for reflexes

Muscle group	Nerve supply	Reflex
Diaphragm	C3, C4, C5	
Shoulder abductors	C5	
Elbow flexors	C5, C6	Biceps jerk
Supinators/pronators	C6	Supinator jerk
Wrist extensors	C6	
Wrist flexors	C7	
Elbow extensors	C7	Triceps jerk
Finger extensors	C7	
Finger flexors	C8	
Intrinsic hand muscles	T1	
Hip flexors	L1, L2	
Hip adductors	L2, L3	
Knee extensors	L3, L4	Knee jerk
Ankle dorsiflexors	L4, L5	
Toe extensors	L5	
Knee flexors	L4, L5, S1	
Ankle plantar flexors	S1, S2	Ankle jerk
Toe flexors	S1, S2	
Anal sphincter	S2, S3, S4	Bulbocavernosus reflex Anal reflex

Table 17.6 Grading of motor power

0	No contraction or movement
1	Minimal movement
2	Active movement, but not against gravity
3	Active movement against gravity
4	Active movement against resistance
5	Active movement against full resistance

Table 17.7 Grading of reflexes

Grade	Description
0	Absent or no response
1+ or +	Sluggish or diminished
2+ or ++	Normal
3+ or +++	Hyperactive without clonus
4+ or ++++	Hyperactive with clonus

Table 17.8 Key areas for sensory examination

1. Shoulder (C4)
2. Inner and outer aspects of the forearms (C6 and T1)
3. Thumbs and little fingers (C6 and C8)
4. Front of both thighs (L2)
5. Medial and lateral aspect of both calves (L4 and L5)
6. Little toes (S1)

Table 17.9 Frankel functional grading of spinal cord injury

Frankel grade	Level of function
Frankel A	Complete paralysis
Frankel B	No voluntary motor, but preserve sensation
Frankel C	Useless motor function
Frankel D	Useful voluntary motor function, but not normal
Frankel E	Normal function

Complete Spinal Cord Transection (Fig. 17.1)

In the acute phase, the classic syndrome of complete spinal cord transection at the high cervical level consists of respiratory insufficiency; quadriplegia with upper and lower extremity areflexia; anaesthesia below the affected level; neurogenic shock (i.e., hypothermia and hypotension without compensatory tachycardia); loss of rectal and bladder sphincter tone; and urinary and bowel retention leading to abdominal distention, ileus, and delayed gastric emptying. This constellation of symptoms is called spinal shock. Horner syndrome (i.e., ipsilateral ptosis, miosis, anhydrosis) is also present with higher

Table 17.10 Techniques to elicit deep tendon reflexes

Reflex	Root value	Test
Biceps	C5, C6	The patient's arm should be partially flexed at the elbow with the palm down Place your thumb or finger firmly on the biceps tendon Strike your finger with the reflex hammer You should feel the response even if you can't see it
Triceps	C6, C7	Support the upper arm and let the patient's forearm hang free Strike the triceps tendon above the elbow with the broad side of the hammer If the patient is sitting or lying down, flex the patient's arm at the elbow & hold it close to the chest
Brachioradialis	C5, C6	Have the patient rest the forearm on the abdomen or lap Strike the radius about 1–2 inches above the wrist Watch for flexion and supination of the forearm
Abdominal	T8, T9, T10, T11, T12	Use a blunt object such as a key or tongue blade Stroke the abdomen lightly on each side in an inward and downward direction above (T8, T9, T10) and below the umbilicus (T10, T11, T12) Note the contraction of the abdominal muscles and deviation of the umbilicus towards the stimulus
Knee	L2, L3, L4	Have the patient sit or lie down with the knee flexed Strike the patellar tendon just below the patella Note contraction of the quadriceps and extension of the knee
Ankle	S1, S2	Dorsiflex the foot at the ankle Strike the Achilles tendon Watch and feel for plantar flexion at the ankle

lesions because of interruption of the descending sympathetic pathways originating from the hypothalamus. Lower cervical level injury spares the respiratory muscles. High thoracic lesions lead to paraparesis instead of quadriparesis, but autonomic symptoms are still marked. In lower thoracic and lumbar/sacral cord lesions, hypotension is not present but urinary and bowel retention are present (Table 17.11).

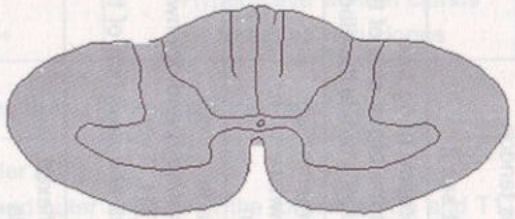


Fig. 17.1 Complete transection of spinal cord

Incomplete Spinal Cord Syndromes

The incomplete SCI syndromes are characterized clinically as follows:

Central cord syndrome *(Fig.17.2)*

This is the most common form of spinal cord syndrome and usually involves cervical cord. It is more common in elderly with pre-existing cervical spondylosis who sustain a hyperextension injury leading to haemorrhage that affects the central part of the spinal cord, destroying the axons of the inner part of the corticospinal tract devoted to the motor control of the hands.

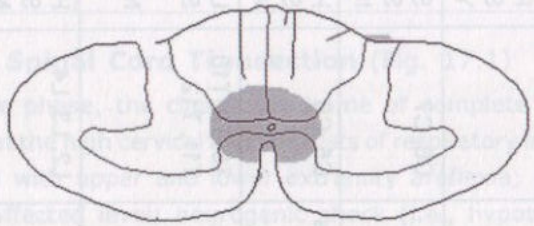


Fig. 17.2 Central cord syndrome

Clinical features. Hand muscles are more affected than lower limb muscles, and distal more than proximal arm weakness. This is because of the arrangement of the fibers system; a central cord injury in the

Table 17.11 Level of complete transection and clinical features

Function	Upper cervical cord injury	Lower cervical cord injury	High thoracic lesions	Lower thoracic & lumbar/ sacral cord lesions
Respiratory insufficiency	+	–	–	–
Motor weakness	Quadriplegia	Quadriplegia	Paraplegia	Paraplegia
Areflexia*	+	+	+	+
Anaesthesia below the affected level	+	+	+	+
Neurogenic shock	+	+	+	+
Loss of rectal and bladder sphincter tone	+	+	+	+
Urinary and bowel retention	+	+	+	+
Delayed gastric emptying	+	+	+	+
Horner syndrome	+	+	–	–

*Areflexia in cervical cord lesions in both upper and lower limbs and in thoracic and lumbar cord lesions only in lower limbs

neck affects the arms more than the legs. This motor weakness may be asociated with patchy sensory loss below the level of the lesion and the most affected sensory modalities are pain and temperature because the lateral spinothalamic tract fibers cross just ventral to the central canal. This is sometimes referred to as dissociated sensory loss and is often present in a capelike distribution. Sacral sensory sparing usually exists in these patients. There may be dysesthesias, especially those in the upper extremities (e.g., sensation of burning in the hands or arms). In these patients bladder dysfunction is common.

Prognosis. Fair.

Anterior cord syndrome *(Fig. 17.3)*

Anterior cord syndrome is the second most common type of spinal cord injury and mechanism of injury is flexion-compression. There is loss of neurologic function in anterior 2/3 of the spinal cord (related to vascular insufficiency—anterior spinal artery infarction).

Clinical features

Spinothalamic—Pain and temperature sensation loss.

Corticospinal—Motor weakness and it is more in the legs than the arms.

Function spared—Posterior columns (position sense, proprioception, vibration, deep pressure).

Prognosis. Worst.

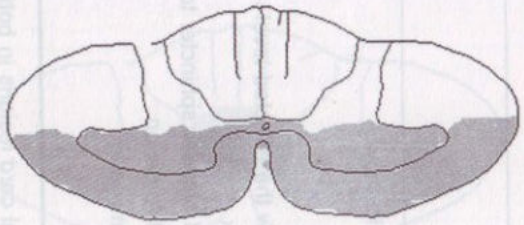


Fig. 17.3 Anterior cord syndrome

Brown-Sequard syndrome *(Fig. 17.4)*

Hemisection of the spinal cord caused by damage to half of the cord.

Clinical features

Ipsilateral—Ipsilateral corticospinal tract weakness (motor weakness) and position/proprioception loss.

Contralateral—Pain and temperature loss (usually two levels below the level of lesion).

Prognosis. Best.

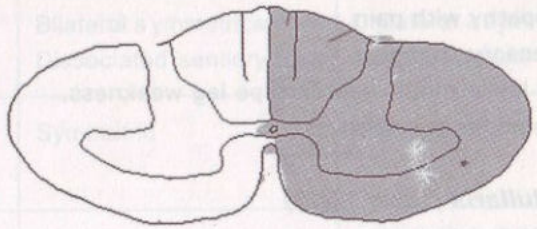


Fig. 17.4 Hemisection of the spinal cord or Brown-Sequard syndrome

Posterior cord syndrome *(Fig. 17.5)*

This type of lesion is very rare.

Clinical features. There is preservation of motor function. Loss of dorsal column function (deep pressure and proprioception) making walking extremely difficult or impossible because of persistent impairment of proprioception.

Prognosis. Good

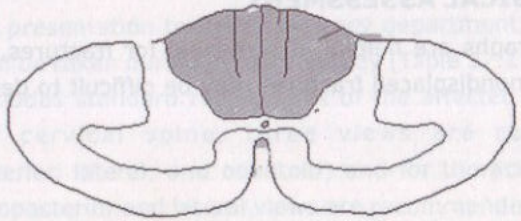


Fig. 17.5 Posterior cord syndrome

Single cervical root lesion

Commonly associated with acute disc protrusion or facet dislocation and often associated with vertebral body rotation.

Most commonly involves C5 or C6 level leading to deltoid and biceps weakness and usually unilateral.

Cauda equina syndrome *(Table 17.12)*

Caused by lumbar vertebral fractures or acute central disc prolapse.

Clinical features

Polyradiculopathy with pain.
Radicular sensory changes.
Asymmetric lower motor neuron type leg weakness.
Bowel and bladder disturbances.

Conus medullaris *(Table 17.12)*

Caused by L1 fracture.

Clinical features

Variable motor and sensory loss in the lower limbs.
Bladder and bowel involvement.

Prognosis. Fair

Spinal cord concussion

Transient neurologic deficit localized to the spinal cord that fully recovers without any apparent structural damage.

RADIOLOGICAL ASSESSMENT

Plain radiographs are helpful in screening for fractures, but hairline fractures or nondisplaced fractures may be difficult to detect.

Table 17.12 Differentiating features between lesions of the conus medullaris and cauda equina

	Conus medullaris	Cauda equina
Spontaneous pain	Not common Less severe Bilateral & symmetric In perineum & thigh	Most prominent symptom Severe radicular type Unilateral & asymmetric In perineum, thighs, legs & back
Sensory deficit	Saddle distribution Bilateral symmetrical Dissociated sensory loss	Saddle distribution Unilateral asymmetrical No dissociation
Motor loss	Symmetric	Asymmetric
Reflexes	Absent ankle jerk	Ankle and knee jerk may be absent
Bladder & bowel	Early & marked	Late & less marked
Trophic changes	Decubitus ulcer common	Less common
Sexual function	Erection and ejaculation impaired	Less impairment

Upon initial presentation to the emergency department, lateral spine X-ray should be taken during primary survey (Table 17.13). Diagnostic imaging includes standard radiographs of the affected region of the spine—for cervical spine, three views are recommended (anteroposterior, lateral, and odontoid) and for thoracic and lumbar spine anteroposterior and lateral views are recommended. Some have recommended a standard trauma series composed of five views: cross-table lateral, Swimmer, oblique, odontoid, and anteroposterior for cervical spine.

Table 17.13 Indications for radiographic evaluation

▪ Patients who exhibit neurologic deficits consistent with a cord lesion ▪ Patients with an altered sensorium from head injury or intoxication ▪ Patients who complain about neck pain or tenderness ▪ Patients who do not complain about neck pain or tenderness but have significant distracting injuries

Remember

- 20% patients with spinal cord injury have no radiological evidence of bony injury.
- A common cause of missed injury is the failure to obtain adequate images.
- Radiographs must adequately depict all vertebrae.
- The cervical spine radiographs must include the C7–T1 junction to be considered adequate.
- If cervical fracture is suspected, visualizing the T1 is important to avoid missing low cervical fractures or subluxation.
- Radiography can miss facet fractures, and dynamic radiographic views (flexion/extension) are often warranted.

Cross-table Lateral View

Approximately 85–90% of cervical spine injuries are evident in lateral view, making it the most useful view from a clinical standpoint. A technically acceptable lateral view shows all 7 vertebral bodies and the cervicothoracic junction. Approach analysis of this view methodically to avoid missing significant pathology.

Check alignment of cervical spine by following three imaginary contour lines (Fig. 17.6):

A. The first line connects the anterior margins of all the vertebrae and is referred to as the anterior contour line.
B. The second line should connect the posterior aspect of all vertebrae in a similar way and is referred to as the posterior contour line.
C. The third line should connect the bases of the spinous processes and is referred to as the spinolaminar contour line.

Each of these lines should form a smooth lordotic curve. Suspect bony or ligamentous injury if disruption is seen in the contour lines. An exception occurs in young children who, because of immature muscular development, may have a benign pseudosubluxation in the upper cervical spine. An imaginary straight line should connect the points bisecting the base of the spinous processes of C1, C2, and C3. In pseudosubluxation, these imaginary points should not be displaced more than 2 mm in front of or behind the straight line.

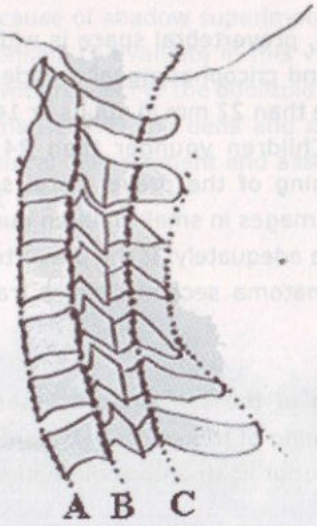


Fig. 17.6 Cervical spine lateral view (sketch)

Check List for Cervical Spine X-ray Lateral View

Check individual vertebrae thoroughly for obvious fracture or changes in bone density. Areas of decreased bone density are seen in patients with osteoporosis, osteomalacia, or osteolytic lesions and may represent weak areas predisposed to injury. Areas of increased bony density may be seen with osteoblastic lesions or may represent compression fractures of an acute nature.

Look for soft tissue changes in predental and prevertebral spaces. The predental space, also known as the atlantodental interval, is the distance between the anterior aspect of the odontoid and the posterior aspect of the anterior arch of C1. This space should be no more than 3 mm in an adult and 5 mm in a child. Suspect transverse ligament disruption if these limits are exceeded.

Prevertebral space extends between the anterior border of the vertebra to the posterior wall of the pharynx in the upper vertebral level (C2–C4) or to the trachea in the lower vertebral level (C6).

At the level of C2, prevertebral space should not exceed 7 mm.

At the level of C3 and C4, it should not exceed 5 mm, or it should be less than half the width of the involved vertebrae.

At the level of C6, prevertebral space is widened by the presence of the oesophagus and cricopharyngeal muscle. At this level, the space should be no more than 22 mm in adults or 14 mm in children younger than 15 years. Children younger than 24 months may exhibit a physiologic widening of the prevertebral space during expiration; therefore, obtain images in small children during inspiration to assess prevertebral space adequately. If the prevertebral space is widened at any level, a haematoma secondary to a fracture is the most likely diagnosis.

Check for fanning of the spinous processes. This is evident as an exaggerated widening of the space between two spinous process tips and suggests posterior ligamentous disruption.

Check for an abrupt change in angulation of greater than 11 degrees at a single interspace. This also suggests bony injury with possible ligamentous involvement.

Swimmer View

Occasionally, it is impossible to fully visualize all 7 cervical vertebrae and, more importantly, the cervicothoracic junction in a true lateral image. If C7 vertebra is not seen then a Swimmer or transaxillary view is necessary.

Oblique View

This view also is considered a laminar view because most pathologic conditions assessed on it manifest with some disruption in the normal overlapping appearance of the vertebral laminae. The normal structural appearance of the laminae is described as shingles on a roof, forming a regular elliptical curve with equal interlaminar spaces. If interlaminar space between two continuous laminae is increased, suspect subluxation of the involved vertebrae. Similarly, if the expected tiling of shingles is disrupted, suspect a unilateral facet dislocation. A posterior laminar fracture should be evident as disruption of the body of a single shingle.

Odontoid View (Fig. 17.7)

This view is used to evaluate an area that is difficult to visualize in the cross-table lateral view because of shadow superimposition. The most important structural relationship to evaluate in this view is alignment of the lateral masses of C1 with respect to the odontoid process. Masses should be bilaterally symmetric with the dens and odontoid process and look for fractures or lateral displacement and assess symmetry of the interspace between C1 and C2.

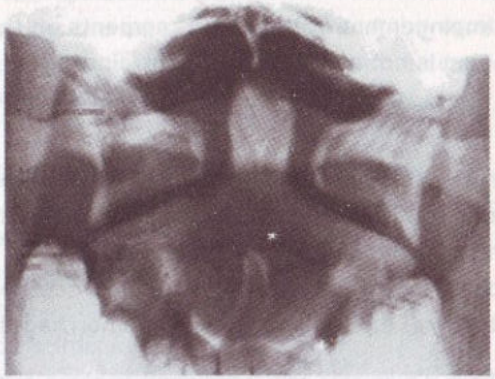


Fig. 17.7 X-ray open mouth view showing odontoid process

Anteroposterior View

This is the least useful view from a clinical standpoint. A straight line should connect the spinous processes bisecting the cervical spine. If this is not seen, consider a rotation injury (i.e., unilateral facet dislocation). Also, consider a Clay Shoveler fracture if a spinous process appears vertically split.

Flexion/Extension Views

Dynamic flexion/extension views are safe and effective for detecting occult ligamentous injury of the cervical spine in the absence of fracture. The negative predictive value of a normal 3-view cervical spine series and flexion/extension views exceeds 99%. The incidence of occult injury in the setting of normal findings on cervical spine radiography and CT scanning is low, so clinical judgment and the mechanism of injury should be used to guide the decision to order flexion/extension views.

CT Scan

Radiography is insensitive to small fractures and also to confirm SCI without radiologic abnormality (SCIWORA), a CT scan documenting the absence of fracture often is necessary. CT imaging is also better for bone definition and is important when radiography shows injury or when an area is poorly visualized. CT imaging can also show soft tissue changes and calcifications. A CT myelogram can be used to determine the degree of impingement of the bony fragments on the thecal scan when MRI imaging is not available or is contraindicated.

MRI

MRI is best for suspected spinal cord lesions, ligamentous injuries, or other nonosseous conditions (extradural spinal haematoma and spinal cord haemorrhage, contusion, and/or oedema). MRI is also the best diagnostic modality for investigation of neurologic deterioration caused by secondary injury (i.e., oedema and/or haemorrhage).

Methylprednisolone

It acts by decreasing oedema and by its anti-inflammatory effect. It protects neuronal membranes by scavenging of O_2 free radicals. Recommended dose of methylprednisolone is IV bolus 30 mg/kg body weight (should be given over 15 min) then followed by IV 5.4 mg/kg body weight/hr for 23 hours. Patient should get methylprednisolone within 3 hours after SCI and always give antacids or proton pump inhibitors with methyl prednisolone.

CHAPTER 18

Cervical Spine Injuries

EPIDEMIOLOGY

20% of deaths from traffic accidents are contributed to or caused by severe cervical spinal cord injury and 80% of those are caused by lesions involving the articulations between the occiput, C1 and C2. Motor vehicle accidents are the most common cause (75%) followed by falls (13%) and sports-related injuries (12%).

Table 18.1 Classification of cervical spine injuries

Hyperflexion injuries
- Anterior subluxation
- Bilateral locked facet joints
- Teardrop fracture
- Spinous process fractures (Clay Shoveller's fracture)

Hyperextension injuries
- Fracture of the anterior or posterior arch of C1
- Anterior-inferior vertebral chip fracture
- Laminar fracture

Axial compression
- Burst fractures
- Fracture of the pedicle of C2

Flexion rotation injuries
- Unilateral facet dislocation

Most frequently fractured vertebral body is C5 and most common level of neurological deficit is at level C5.

CLASSIFICATION OF CERVICAL SPINE FRACTURES

Cervical spine fractures can be classified according to the mechanism of injury or level of injuries (Table 18.1).

GENERAL PRINCIPLES OF MANAGEMENT AND IMAGING

Cervical spine injuries should be dealt with carefully and guidelines for evaluation and management are discussed in Chapter 16 & 17. Here are some guidelines to assess cervical spine X-rays (Table 18.2) and instability in cervical spine following trauma (Table 18.3).

Table 18.2 Radiological assessment of cervical spine

- Lateral cervical spine X-ray should be taken during primary survey
- Should ensure that the junction between C7 and T1 is seen
- If unable to see the C7/T1 junction consider a 'Swimmer's view'

On lateral cervical spine films assess

- Anterior vertebral alignment
- Posterior vertebral alignment
- Posterior facet joint margins
- Anterior border of spinous processes
- Posterior border of spinous processes
- Integrity of vertebral bodies, laminae, pedicles and arches
- Prevertebral space
- Interspinous gaps
- Anterioposterior and odontoid peg views should be taken during secondary survey

Remember 20% patients with spinal cord injury have no radiological evidence of body injury

Table 18.3 Radiological signs of cervical spine instability

- Compression of vertebral body > 25%
- Kyphotic angle of > 10°
- Facet joint widening
- Teardrop fracture
- Base of odontoid peg fracture
- Atlanto-occipital dislocation
- Atlanto-axial gap > 3 mm

SPECIFIC CONSIDERATIONS

Flexion Injuries

Fig. 18.1 X-ray cervical spine showing all seven vertebrae

Simple wedge fracture

With a pure flexion injury, a longitudinal pull is exerted on the nuchal ligament complex that, because of its strength, usually remains intact and the anterior vertebral body bears most of the force, sustaining simple wedge compression anteriorly without any posterior disruption. Radiographically, the anterior border of the vertebral body has diminished height and increased concavity along with increased density due to bony impaction. The prevertebral soft tissues are swollen.

The posterior column remains intact, making this a stable fracture that requires only use of a cervical orthosis for treatment.

Flexion teardrop fracture

A flexion teardrop fracture occurs when flexion of the spine, along with vertical axial compression, causes a fracture of the anteroinferior aspect of the vertebral body. This fragment is displaced anteriorly and resembles a teardrop. For this fragment to be produced, significant posterior ligamentous disruption must occur. Since the fragment displaces anteriorly, a significant degree of anterior ligamentous disruption exists. This injury involves disruption of all three columns, making this an extremely unstable fracture that frequently is associated with spinal cord injury. Initial management is application of traction with cervical tongs followed by fixation.

Anterior subluxation *(Fig. 18.2 & 18.3)*

Anterior subluxation in the cervical spine occurs when posterior ligamentous complexes (nuchal ligament, capsular ligaments, ligamenta flava, posterior longitudinal ligament) rupture.

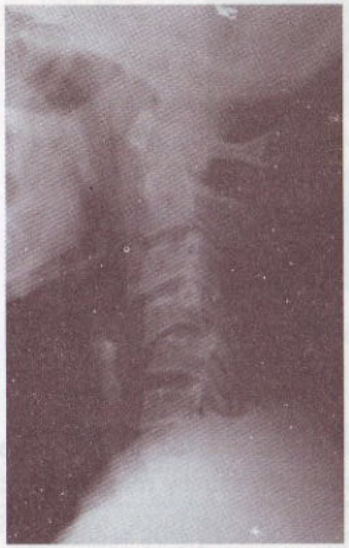


Fig. 18.2 X-ray cervical spine showing C4 anterior subluxation over C5

Fig. 18.3 MRI cervical spine showing C4 subluxation over C5 with minimal compression of cord

Fig. 18.4 Postoperative X-ray cervical spine showing C4-C5 fixation with plate and screws

The anterior longitudinal ligament remains intact. No associated bony injury is seen. Radiographically, the lateral view shows widening of interspinous processes, and anterior and posterior contour lines are disrupted in flexion views. Since the anterior columns remain intact, this fracture is considered mechanically stable by definition. Anterior subluxation is rarely associated with neurologic sequelae. Nevertheless, most authorities approach this injury as if it were potentially unstable because of the significant displacement that can occur with flexion, and very rare cases have associated neurologic deficit.

Bilateral facet dislocation

Bilateral facet dislocation is an extreme form of anterior subluxation that occurs when a significant degree of flexion and anterior subluxation causes ligamentous disruption to extend anteriorly, which causes significant anterior displacement of the spine at the level of injury. This injury involves the annulus fibrosus, anterior longitudinal ligament, and posterior ligamentous complex. At the level of injury, i.e., the upper vertebrae, inferior articulating facets pass superior and anterior to the superior articulating facets of the lower involved vertebrae because of extreme flexion of the spine. This is an extremely unstable

condition and is associated with a high prevalence of spinal cord injuries. A significant number of bilateral facet dislocations are accompanied by disc herniation and it may further damage the spinal cord. Initial management is closed reduction and traction with cervical tongs followed by fixation.

Clay Shoveler fracture

Abrupt flexion of the neck, combined with a heavy upper body and lower neck muscular contraction, results in an oblique fracture of the base of the spinous process, which is avulsed by the intact and powerful supraspinous ligament. This type of fracture can also occur with direct blows to the spinous process or with trauma to the occiput that causes forced flexion of the neck. Fracture commonly is observed in a lateral view, since the avulsed fragment is readily evident. It may also be seen in the anteroposterior view as a vertically split appearance of the spinous process. This type of injury commonly occurs in lower cervical vertebrae; therefore, visualization of the C7–T1 junction in the lateral view is imperative. Since this injury involves only the spinous process, this fracture is considered stable, and it is not associated with neurologic impairment. Management involves only cervical immobilization in a collar.

Flexion-rotation injury

Common injuries associated with a flexion-rotation mechanism include unilateral facet dislocation and rotary atlantoaxial dislocation.

Unilateral facet dislocation

Unilateral facet dislocation occurs when flexion, along with rotation, forces one inferior articular facet of an upper vertebra to pass superior and anterior to the superior articular facet of a lower vertebra, coming to rest in the intervertebral foramen. Vertebrae are locked in place, making this injury stable. Radiographically, the lateral view shows an anterior displacement of the spine at the involved level of less than one half the diameter of the vertebral body. This is in contrast to the greater displacement seen with a bilateral facet dislocation, as discussed above. The anteroposterior view is useful in diagnosis of unilateral dislocation because it shows a disruption in the line connecting the spinous processes at the level of the dislocation. The oblique view will show a disruption of the typical shingles appearance at the level of the involved vertebra. The dislocated superior articulating facet of the lower vertebra is seen projecting within the neural foramina. The injury seldom is associated with neurologic deficits. The initial management is cervical traction to attempt closed reduction.

Rotary atlantoaxial dislocation

This injury is a specific type of unilateral facet dislocation at atlantoaxial level. Radiographically, the odontoid view will show asymmetry of the lateral masses of C1 with respect to the dens along with unilateral magnification of a lateral mass of C1 (wink sign). To confirm true dislocation, basilar skull structures (jugular foramina) should appear symmetric in the presence of the findings described above. This injury is considered unstable because of its location.

Extension Injury

Common injuries associated with an extension mechanism include hangman fracture, extension teardrop fracture, fracture of the posterior arch of C1 (posterior neural arch fracture of C1), and posterior atlantoaxial dislocation.

Hangman fracture (traumatic spondylolisthesis of C2)

It is the type of fracture secondary to judicial hanging. It is a fracture-dislocation of C2 and more precisely, it is a bilateral pedicle fracture of C2, along with distraction of C2 from C3 secondary to complete disruption of the disc and ligaments between C2 and C3. The mechanism of injury is a combination of hyperextension and distraction with varying degrees of axial compression and lateral flexion.

Extension teardrop fracture

As with flexion teardrop fracture, extension teardrop fracture also manifests with a displaced anteroinferior bony fragment. This fracture occurs when the anterior longitudinal ligament pulls fragment away from the inferior aspect of the vertebra because of sudden hyper-extension. The fragment is a true avulsion, in contrast to the flexion teardrop fracture in which the fragment is produced by compression of the anterior vertebral aspect due to hyperflexion. This type of fracture is common after diving accidents and tends to occur in lower cervical levels. It also may be associated with the central cord syndrome due to buckling of the ligamenta flava into spinal canal during the hyper-

extension phase of the injury. This injury is stable in flexion but highly unstable in extension. Initial management is avoidance of iatrogenic extension and cervical traction with tongs followed by fixation.

Fracture of the posterior arch of C1 (posterior neural arch fracture)

This fracture occurs when the head is hyperextended and the posterior neural arch of C1 is compressed between the occiput and the strong and prominent spinous process of C2, causing the weak posterior arch of C1 to fracture. Radiographically, the lateral projection shows a fracture line through the posterior neural arch. The odontoid view fails to show any displacement of the lateral masses of C1 with respect to the articular pillars of C2, a finding that distinguishes this fracture from a Jefferson fracture. The transverse ligament and the anterior arch of C1 are not involved, making this fracture stable. Initial management involves the differentiation of this benign fracture from a Jefferson fracture. Once this is accomplished, only use of a cervical colllar is required.

Vertical (axial) Compression Injury

Common injuries associated with a vertical compression mechanism include Jefferson fracture (burst fracture of the ring of C1), burst fracture (dispersion, axial loading), atlas fracture, and isolated fracture of the lateral mass of C1 (pillar fracture).

Jefferson fracture (burst fracture of the ring of C1)

This fracture is caused by a compressive downward force that is transmitted evenly through the occipital condyles to the superior articular surfaces of the lateral masses of C1. The process displaces the masses laterally and causes fractures of the anterior and posterior arches, along with possible disruption of the transverse ligament. Radiographically, it is characterized by bilateral lateral displacement of the articular masses of C1. The odontoid view shows unilateral or bilateral displacement of the lateral masses of C1 with respect to the articular pillars of C2; this finding differentiates it from a simple fracture of the posterior neural arch of C1. The lateral projection usually reveals a striking amount of prevertebral soft tissue oedema.

Burst fracture of the vertebral body

When downward compressive force is transmitted to lower levels in the cervical spine, the body of the cervical vertebra can shatter outward, causing a burst fracture. This fracture involves disruption of the anterior and middle columns, with a variable degree of posterior protrusion of the latter. Radiographically, this fracture is evidenced by a vertical fracture line in the frontal projection and by comminution and protrusion of the vertebral body anteriorly and posteriorly with respect to the contiguous vertebrae in the lateral view. Middle column posterior protrusion may extend into the spinal canal and can be associated with anterior cord syndrome. Burst fractures always require an axial CT scan or MRI to document amount of middle column retropulsion. Initially manage burst fractures with a loss in height of more than 25%, retropulsion, or neurologic deficit by applying traction with cervical tongs. When none of those problems exist, the fracture is considered stable.

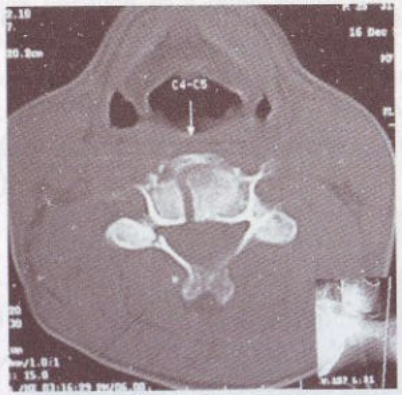


Fig. 18.5 CT scan cervical spine showing burst fracture of vertebral body

Atlas (C1) fractures

Isolated atlas injuries are more common (56%) followed by multiple fractures of the atlas ring (31%) and unilateral ring fractures (13%). Neurological injury following isolated atlas fracture is rare. Atlas fractures result from impaction of the occipital condyles on the atlas, causing single or multiple fractures around the ring. Jefferson fracture (burst fracture of the ring of C1) is tne most significant type of atlas

fracture from a clinical standpoint because it is associated with neurologic impairment (discussed above). Initial management of types I, II, and III atlas fractures consists of placement of a cervical orthosis. Type IV fracture, or Jefferson fracture, is managed with cervical traction.

C2 lateral mass fractures

This type of injury is extremely rare and if found, other C-spine pathology must be sought. The mechanism of this type of fracture is axial compression with concomitant lateral bending. The isolated fracture may present with high neck pain and a normal neurological examination. Radiographic findings include impaction of the C2 component of the atlantoaxial articulation surface, asymmetry of C2 lateral mass height, and lateral tilting of the arch of C1.

Multiple or Complex Injuries

Odontoid process fractures

The three types of odontoid process fractures are classified (Anderson and D lonzo) based on the anatomic level at which the fracture occurs (Fig. 18.6).

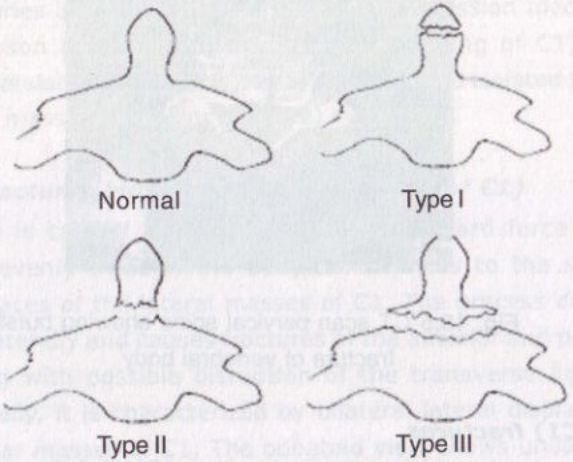


Fig. 18.6 Types of odontoid fracture

Incidence of odontoid fractures approaches 15% of all C-spine fractures and most common causes are motor vehicular accidents and falls. When an odontoid fracture is suspected, it is important to rule out concomitant associated C-spine injuries. For example, C1 anterior ring fractures are not an uncommon finding, and a prevertebral soft-tissue shadow of more than 10 mm on plain films is highly suggestive of such a fracture.

Type I odontoid fracture (~ 5%) is an avulsion of the tip of the dens at the insertion site of the alar ligament. Although a type I fracture is mechanically stable, it often is seen in association with atlanto-occipital dislocation and must be ruled out because it is life threatening.

Type II fractures (60%) occur at the base of the dens and are the most common odontoid fractures. This type is associated with a high prevalence of nonunion because of the limited vascular supply and a small area of cancellous bone (Figure 18.7 to 18.9).

Type III odontoid fracture (30%) occurs when the fracture line extends into the body of the axis. Nonunion is not a major problem with these injuries because of a good blood supply and the greater amount of cancellous bone. With types II and III fractures, the fractured segment may be displaced anteriorly, laterally, or posteriorly. Since posterior displacement of segment is more common, the prevalence of spinal cord injury is as high as 10% with these fractures.

Fig. 18.7 X-ray cervical spine showing type-II odontoid fracture

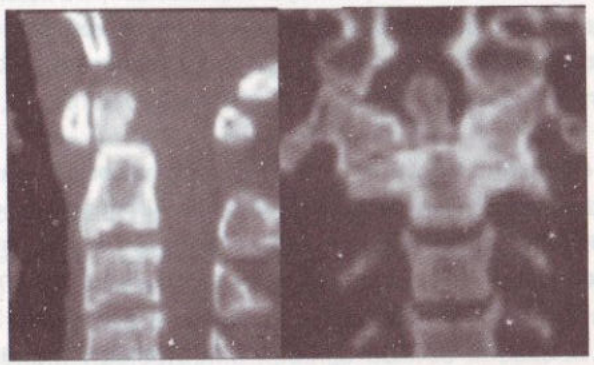


Fig. 18.8 CT scan sagittal and coronal reconstruction showing type-II odontoid fracture

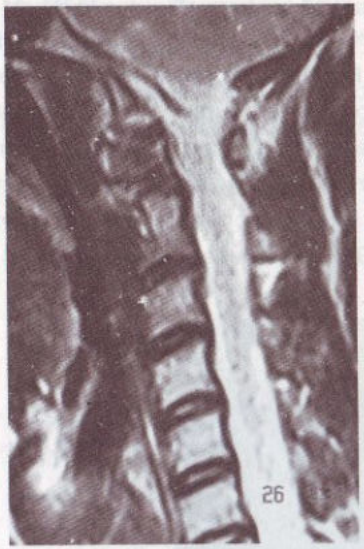


Fig. 18.9 MRI of type -II odontoid fracture

Fig. 18.10 Sublaminar wiring for type-II odontoid fracture

Management. Management of a type I dens fracture is by use of a cervical orthosis and types II and III fractures by applying traction with cervical tongs. As the chances of non-union are very high with type II fractures, they may need some type of fixation and the techniques for C1/C2 fusion include (Fig. 18.10):

- Sublaminar wiring
- Odontoid screw
- Transarticular screws

Atlantoaxial subluxation

When flexion occurs without a lateral or rotatory component at the upper cervical level, it can cause an anterior dislocation at the atlantoaxial joint if the transverse ligament is disrupted and because this joint is near the skull, shearing forces also play a part in the mechanism causing this injury, as the skull grinds the C1-C2 complex in flexion. Since the transverse ligament is the main stabilizing force of the atlantoaxial joint, this injury is unstable. Neurologic injury may occur from cord compression between the odontoid and posterior arch of C1. Radiographically, this injury is suspected if the predental space is more than 3 mm (5 mm in children). CT scan is required to confirm the diagnosis. Tnese injuries may require fusion of C1 and C2 for definitive management.

Atlanto-occipital dislocation

When severe flexion or extension exists at the upper cervical level, atlanto-occipital dislocation may occur. Atlanto-occipital dislocation involves complete disruption of all ligamentous relationships between the occiput and the atlas.

Classification

1. Anterior displacement of the cranium with respect to the atlas.
2. Longitudinal distraction with separation of skull from cervical spine.
3. Posterior displacement of the cranium with respect to the atlas.

Death usually occurs immediately from stretching of the brainstem, which causes respiratory arrest.

Clinical features. Traumatic atlanto-occipital dislocation is a priori an unstable injury, and is usually fatal at the accident scene. Patients may be neurologically intact but this is rare. Usually they present with a number of clinical patterns (Box).

- Dead (bulbar-cervical dissociation)
- Lower cranial nerve deficits long tract signs
- Worsening of neurological deficit following application of traction
- Subarachnoid haemorrhage, especially in the posterior fossa

Imaging. Separation of atlas from occipital condyles is the hallmark of OA dislocation. Significant displacement of the odontoid from the basion also occurs. Plain X-rays show a retropharyngeal haematoma.

Normal distance from the basion to the odontoid is about 5 mm (4–11 mm). Normal translation between the clivus and arch of C1 is 1 mm. CT, MRI, and a posterior circulation angiogram may all be necessary.

Management. Halo immobilization is required for 10–12 weeks. If instability persists, need internal fixation with posterior occipito-cervical fusion. Cervical traction is absolutely contraindicated, since further stretching of the brainstem can occur.

Occipital condyle fracture

Occipital condyle fractures are caused by a combination of vertical compression and lateral bending. Avulsion of the condylar process or a comminuted compression fracture may occur secondary to this mechanism. They are of three types (communited, extension of basilar skull fracture and avulsion of condyle into foramen magnum). These fractures are associated with significant head trauma and usually are accompanied by cranial nerve deficits. Radiographically, they are difficult to delineate, and axial CT may be required to identify them.

Management

1. Comminuted—Are stable and treated with rigid collar.
2. Extension of basilar skull fracture—Are stable and treated with rigid collar.
3. Avulsion of condyle into foramen magnum—Fragment usually attached to alar ligament and unstable and requires halo immobilization.

CHAPTER 19

Thoracolumbar Injuries

Anatomical characteristics of dorsolumbar region make it more prone for spine and spinal cord injuries (Box).

- Longest component of the spinal cord
- Spinal canal and vertebral bodies are proportionately smaller
- Vascular supply is more tentative
- Few collateral vessels
- Small anterior spinal arteries
- Small radicular arteries

CLASSIFICATION

Based on the mechanism of injury, fractures of the thoracolumbar spine can be classified into four groups. The mechanism of injury is used interchangeably with the name of the fracture. These major fractures are discussed in escalating order of severity.

Flexion-compression Mechanism (wedge or compression fracture)

This mechanism usually results in an anterior wedge compression fracture (Fig. 19.1, 19.2 & 19.3). As the name implies, the anterior column is compressed, with varying degrees of middle and posterior column insult. Ferguson and Allen further classified these lesions into three distinct patterns of injury.

Pattern-I

The first pattern involves anterior column failure while the middle and posterior columns remain intact. Imaging studies demonstrate wedging of the anterior component of the vertebral bodies. Loss of anterior vertebral body height is usually less than 50%. This is a stable fracture.

Pattern-II

The second pattern involves both anterior column failure and posterior column ligamentous failure. Imaging studies demonstrate anterior wedging and may indicate increased interspinous distance. Anterior wedging can produce a loss of vertebral body height greater than 50%. This has an increased possibility of being an unstable injury.

Pattern-III

The third pattern involves failure of all three columns. Imaging studies demonstrate not only anterior wedging, but also varying degrees of posterior vertebral body disruption. Additionally, the possibility exists for cord, nerve root, or vascular injury from free-floating fracture fragments dislodged in the spinal canal. This is an unstable fracture.

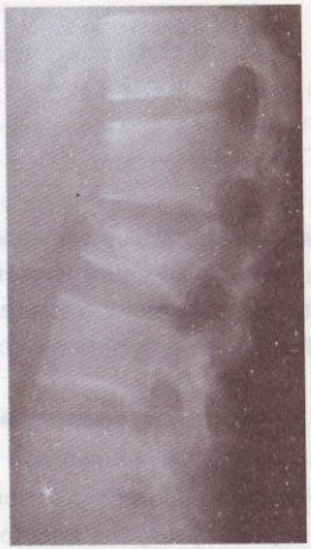


Fig. 19.1 X-ray dorso-lumbar spine showing wedge fracture of L-1 body

Fig. 19.2 X-ray dorsolumbar spine showing wedge fracture of L-1 body

Fig. 19.3 MRI of L-1 wedge fracture (sagittal section) showing compression of thecal sac

Axial-compression Mechanism

This mechanism results in an iniury called a burst fracture, and the pattern involves failure of both the anterior and middle columns resulting in the loss of height of the vertebral body. There are five subtypes and each is dependent on concomitant forces, namely rotation, extension, and flexion (Box).

- Fracture of both endplates
- Fracture of the superior endplate (most common)
- Fracture of the inferior endplate
- Burst rotation fracture
- Burst lateral flexion fracture

McAfee classified burst fractures based on the constitution of the posterior column (stable or unstable). In stable burst fractures, the posterior column is intact; in unstable burst fractures, the posterior column has sustained significant insult. Unstable fractures may have posterior element displacement and/or vertebral body or facet dislocation or subluxation. As with a severe wedge fracture, the possibility exists for a cord, nerve root, or vascular injury from posterior

displacement of fracture fragments into the canal. Rate of neurologic sequelae in these patients could be as high as 50%. Imaging studies of both stable and unstable burst fractures demonstrate loss of vertebral body height and MRI studies will further identify the possibility of canal impingement, which will require decompressive surgery.

Flexion-distraction Mechanism

This mechanism results in an injury called a Chance (or seatbelt) fracture. This pattern involves failure of the posterior column with injury to ligamentous components, bony components, or both. The pathophysiology of this injury pattern is dependent on the axis of flexion. Several subtypes of this type fracture are described and each is dependent on the axis of flexion and on the number and degree of column failure:

The classic Chance fracture has its axis of flexion anterior to the anterior longitudinal ligament; this results in a horizontal fracture through the posterior and middle column bony elements along with disruption of the supraspinous ligament. This is considered a stable fracture. Imaging studies show an increase in the interspinous distance and possible horizontal fracture lines through the pedicles, transverse processes, and pars interarticularis.

The flexion-distraction subtype that has its axis of flexion posterior to the anterior longitudinal ligament in addition to the previously mentioned radiographic findings; it is also associated with an anterior wedge fracture. Because all three columns are involved, this is considered an unstable injury.

Rotational Fracture-dislocation Mechanism

The precise mechanism of this fracture is a combination of lateral flexion and rotation with or without a component of posterior-anteriorly directed force. The resultant injury pattern is failure of both the posterior and middle columns with varying degrees of anterior column insult. The rotational force is responsible for disruption of the posterior ligaments and articular facet. With sufficient rotational force, the upper vertebral body rotates and carries the superior portion of the lower vertebral body along with it. This causes the radiographic "slice" appearance sometimes seen with these types of injuries. Subtypes of this type of fracture-dislocations are flexion-rotation, flexion-distraction and shear injuries.

The flexion-rotation injury pattern results in failure of both the middle and posterior columns along with compression of the anterior column. Imaging studies may demonstrate vertebral body subluxation or dislocation, increased interspinous distance, and an anterior wedge fracture. The shear (sagittal slice) injury pattern results in a 3-column failure. The combined rotational and posterior-to-anterior force vectors result in vertebral body rotation and annexation of the superior portion of the adjacent and more caudal vertebral body. Imaging studies demonstrate both the nature of the fracture and dislocation.

Minor Fractures

Minor fractures include fractures of the transverse processes of the vertebrae, spinous processes, and pars interarticularis. Minor fractures do not usually result in associated neurologic compromise and are considered mechanically stable. However, because of the large forces required to cause these fractures, associated abdominal injuries may occur.

SURGICAL THERAPY

Goal of Treatment

The goals of operative treatment are to decompress the spinal cord canal and to stabilize the disrupted vertebral column.

Operative Approaches

Three basic approaches are used for surgical management of the thoracolumbar spine (Box). Selection of the best approach is guided by the anatomy of the fracture and the location of spinal canal encroachment and the need for stabilization procedures.

- Posterior a pproach
- Posterolateral a pproach
- Anterior approach

Posterior approach

The posterior approach with a midline incision and a laminectomy allows for access to the posterior elements, although it does not permit

access to the vertebral bodies and, as a result, is not commonly used. Spinal cord compression as a result of isolated fractures of the posterior elements is not very common. Spinal canal compromise is more frequent when the vertebral bodies and anterior elements are involved. The posterior approach is useful for stabilization procedures that involve fixation of the posterior bony elements (Fig. 19.4). The posterior approach is used when early mobilization is considered and decompression of the spinal canal is not a major consideration.

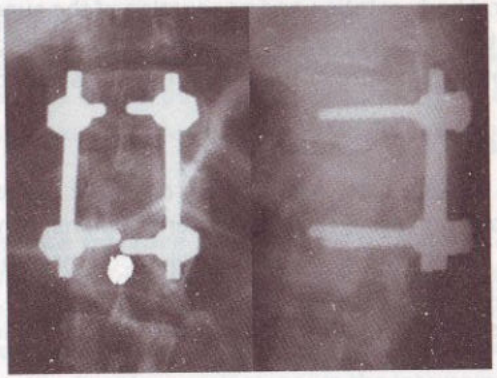


Fig. 19.4 Fixation of dorsolumbar spine with transpedicular screws and rods

Posterolateral technique

The posterolateral technique improves access to the vertebral bodies, although access is still limited. Decompression of ventral impingement of the canal is technically difficult using this approach. It is useful when only a limited exposure of the ventral elements is required. It may be combined with a posterior stabilization procedure when limited ventral exposure is needed. The approach to the high thoracic segments is technically difficult. This technique is often used for high thoracic fractures such as T1 through T4.

Anterior approach

The anterior approach allows access to the vertebral bodies at multiple levels. Transthoracic exposure is required in order to access the vertebral bodies down to L2. Lower fractures require a transabdominal-retroperitoneal exposure. It is most useful for decompression of injuries and spinal canal compromise caused by vertebral body fractures (burst fracture, sagittal slice fracture, and severe compression fractures). When anterior vertebral bodies are resected and replaced with autologous bone graft, it will delay the patient mobilization.

Procedures for Spine Stabilization

The three basic types of stabilization procedures are shown in Box and each has different advantages and disadvantages and following removal of vertebral body a bone graft or cage can be placed in the defect.

- Posterior lumbar interspinous fusion
- Posterior rods
- Z-plate anterior thoracolumbar plating system

Posterior lumbar interspinous fusion is the least-invasive method and involves the use of screws to obtain stability and promote fusion. Most patients have good results with this technique. It can be used effectively for isolated or relatively stable fractures.

Posterior rods require extensive exposure and are effective in stabilizing multiple fractures or unstable fractures. The use of rods prevents further deformity and deterioration. The rods are attached with pedicle screws, stainless steel wires, clips, and clamps to achieve a stable construct.

The Z-plate anterior thoracolumbar plating system has been used for the treatment of burst fractures. Surgery is performed for neurologic deficits, deformity, progressive kyphosis, and late pain. Ghanayem and Zdeblick reported good success with this form of anterior arthrodesis.

CHAPTER 20

Paediatric Injuries

INTRODUCTION

Present knowledge about the fundamental differences between paediatric and adult spinal column and spinal cord injuries remains incomplete and it has received increasing attention over the last two decades. The overall incidence of paediatric spine trauma within the overall population of spinal injuries varies between 1% and 11%. Males are more frequently affected than females.

ANATOMY AND BIOMECHANICAL CONSIDERATIONS

There are significant anatomical and biomechanical differences between the paediatric spine and its adult counterpart. The infant spine is defined as that between 0 and 2 years of age and between the ages of 2 and 10 it starts developing the characterisitics of adult spine. Age related maturation that occurs in the upper paediatric cervical spine is usually completed by approximately age 10 and the maturation of the lower cervical spine occurs by approximately age 14.

Characteristics of Paediatric Spine

Infant spine

- Tremendous mobility and elasticity due to underdevelopment of the neck muscles
- Incompletely calcified, wedge-shaped vertebrae, and shallow, horizontally oriented spine (facet) joints.
- The relatively large size of the head with respect to the torso in young patients, increases the likelihood of cervical spine injuries, especially between the skull and first cervical vertebrae.

Between the ages of 2 and 10

- Tremendous changes occur in the spinal column.
- Muscles and ligaments strengthen, bones grow and reach a mature shape and size, and areas of cartilage and soft bone are replaced with normal calcified bone.
- The body habitus changes so that the head is smaller in proportion to the torso.
- Shift of focus of injury from the upper cervical spine (skull-C1-C2) to the lower cervical spine (C5-C6).

The elasticity of the paediatric spinal column probably allows some protection against spinal cord trauma that might cause fracture in older patients. This mobility and elasticity in the infant spine explains the relatively low incidence of spinal column injuries and the proportionately high incidence of spinal cord injuries without radiographic abnormalities (discussed later).

MECHANISMS OF INJURY

There are numerous mechanisms responsible for the patterns of paediatric spinal cord and spinal column injury including flexion, extension, rotation, axial (top), loading and distraction (pulling). Compression of the spinal cord from blood clots, fractured bones, bending or buckling of ligaments, and angulation of the spinal column will lead to ischaemic damage. Underlying diseases, either congenital or acquired, will also contribute to the risk of spinal cord injury (e.g., os odontoideum, Down syndrome, Chiari malformations, congenital bone abnormalities, rheumatoid arthritis, ankylosing spondylitis, and other underlying infections or tumours may all play a part in the predisposition to spinal column or spinal cord injury). The actual type of injury responsible for the mechanism varies according to the age of the patient and the youngest age group patients (0 to 10 years) have a high incidence of falls and pedestrian/automobile accidents, older children have a higher incidence of motor vehicle accidents, motorcycle accidents and sports-related injuries.

SPECIFIC INJURIES

Young children tend to sustain soft tissue injuries without incurring true fractures, a finding that reflects hypermobility and skeletal immaturity.

Most of the spinal injuries in the first decade of life affect the upper segments of the cervical spinal column. Cervical sprain is perhaps the most common type of spinal injury, but has been poorly documented due to its trivial status. Serious ligamentous injuries and dislocations have a higher incidence in the upper cervical spine in younger children. Dislocation between the skull and the first cervical vertebra is typically due to injury to the strong ligaments that normally keep this joint intact. Injuries in this area usually result in extreme instability at the craniocervical junction. Unfortunately, many of these injuries are fatal and their overall incidence is probably underreported. Young children are also prone to dislocation without fracture. A higher rate of persistent instability can occur in comparison to instability associated with fracture dislocation injuries. Young children typically suffer growth plate fractures and separations. This mechanism is thought to be the aetiologic factor behind os odontoideum, a condition where part of the second cervical vertebra is separate from the vertebral body. This separation leads to chronic instability and an increased risk of spinal cord injury.

Spinal Cord Injury Without Radiographic Abnormality (SCIWORA)

SCIWORA is defined as the occurrence of a spinal cord injury despite normal plain radiographic studies including normal flexion/extension films, CT scan. Incidence ranges from 5% to 70% (average 20%). SCIWORA is common among children, uncommon in adolescents and rare among adults and 2/3 of the cases occur in patients 8 years or younger. Cervical and thoracic spinal levels are injured with almost equal frequency and lumbar levels are rarely involved. Younger patients (0 to 8 years) have a higher proportion of complete neurological injuries than adolescents.

Pathophysiology

SCIWORA is due to the ligamentous flexibility and elasticity of the immature spine. A young child's vertebral column can withstand up to two inches of stretch without disruption and in contrast, the spinal cord ruptures only after 1/4 inch of stretching. This mismatching of elasticity response between the spinal column and spinal cord is the major factor contributing to the high incidence of SCIWORA injuries in young children.

Clinical features and investigations

It depends on the severity of lesion and completeness of injury (neck pain, weakness, sensory loss etc.). In all cases of suspected SCIWORA injury, an MRI should be performed to rule out treatable lesions that may not be identified on plain films. Examples of such lesions include haematomas or hidden ligamentous instability not shown on other studies and delayed onset of neurological deficits may predispose to a worse outcome.

Management

Maintain patients in external braces such as a stiff cervical collar for several months (at least one to two months) in order to prevent further injury. It is important to understand that once a SCIWORA injury is diagnosed, the child is at increased risk for recurrence of this episode and recurrent injuries are typically more severe than initial injuries and may have permanent sequelae.

Birth Injuries

Spinal cord injury due to birth related trauma is probably under diagnosed and underreported. This is most likely due to the lack of radiographic findings seen in most instances. Typically, the upper cervical spine or cervicothoracic junction is affected. However, any level of the spinal cord can be involved and involvement in multiple levels is not uncommon. Two-thirds of all birth injuries accompany breech presentation and 1/3 occur with cephalic presentation or transverse lie. There have been a wide variety of factors implicated in birth related spinal cord trauma including mechanical repositioning, breech presentation and forceps extraction. The mortality for birth related spinal cord injury is high and survivors may have a poor prognosis. Improved prenatal monitoring and obstetrical techniques will help to reduce these injuries.

TREATMENT OF PAEDIATRIC SPINAL CORD INJURY

The principles involved in treatment of paediatric spinal cord injury are similar to those described for adults.

CHAPTER 21

Complications of Spinal Injuries

The patient with spinal cord injuries experiences different type of complications and characteristics associated with aging. These complications commonly include pain, urinary tract infections and bowel and bladder disturbances apart from motor and sensory deficits.

Pain

Upper extremity pain is a common problem associated with spinal cord injury and is most often due to either peripheral nerve entrapments or overuse syndromes. The most common overuse syndromes causing pain in the upper extremities are degenerative joint diseases, rotator cuff tears, rotator cuff tendinitis, subacromial bursitis, and capsulitis.

Scoliosis

Nearly all paediatric patients with SCI experience scoliosis (97%). In adults with SCI, just slightly less than 50% develop scoliosis.

Osteoporosis

Osteoporosis occurs uniformly in individuals with SCI with bone loss beginning immediately after the injury. There is a continual linear loss of bone that is a function of time since the occurrence of SCI. Individuals with a Brown-Sequard-type SCI have been shown to have greater bone loss in the paretic extremity as compared to the stronger extremity.

Thrombo-embolic Disease

Thrombo-embolic disease is common following SCI. Approximately 40% of patients without prophylaxis develop DVT during the acute phase. The risk of death from pulmonary embolism during the first year following SCI is more than 200 times that for the general population. DVT most commonly occurs in the weeks following SCI, with a much lower risk in persons with chronic injury. Classic symptoms of DVT, such as calf tenderness, may be lacking, owing to sensory loss. DVT can present with fever of unknown origin, and pulmonary embolism can result in sudden death. A prophylactic strategy can address venous stasis and hypercoagulability. Pneumatic compression devices can be used for the first 2 weeks, followed by use of a compression hose. Unfractionated heparin (5000 U SC q12h) or a low molecular weight heparin (30 mg SC q12h), such as enoxaparin, can be administered for 2–3 months following injury.

Soft Tissue Changes

Soft tissue changes occur as a result of aging with SCI. The areas over weight-bearing surfaces (the buttocks) experience a loss or thinning of the subcutaneous tissues. This results in thinning of the skin and a loss of elasticity. These effects result in skin that is subject to breakdown and is harder to heal once a pressure sore or ulcer has developed. The rate of decubitus ulcers increases with time after the onset of SCI. A loss of lean muscle mass also occurs with time.

Cardiovascular Disease

The incidence of cardiovascular disease among individuals with SCI is over 200% higher than what is expected in age and gender-matched control population. Diabetes has been reported to be 4 times more common in men with SCI. Hypertension is nearly twice as common in individuals with paraplegia as in able-bodied controls. Reduced exercise tolerance because of ineffective distribution of oxygenated blood. Venous return to the heart is limited because of decreased sympathetic tone and a decreased muscular pumping action of the lower extremities, leading to pooling.

Urologic Complications

Patients with spinal cord injuries most of the time either require indwelling catheter or self intermittent catheterization. This will increase the chances of infection and with their sequelae. Also, patients with incomplete injuries are more prone for stone formation particularly in the bladder.

Gastrointestinal Complications

Gastrointestinal complications are common both with acute and chronic SCI. Following chronic SCI, colonic compliance and motility decrease. Basal colonic myoelectrical activity is higher in persons with SCI than in controls and problems associated with GI tract dysfunction increase with age after SCI. Patients with spinal cord injuries are prone to develop haemorrhoids, abdominal distention, autonomic dysreflexia related to the GI tract, and difficulty with bowel evacuation.

Pulmonary Function

Declining pulmonary function can be a result of restrictive disease, obstructive disease, or a combination of the two. In SCI, restrictive lung disease occurs as a result of respiratory muscle paralysis. The development of kyphosis, scoliosis, or increasing spasticity can cause further restrictive disease as the individual with SCI ages. The higher the level of SCI, the greater the restrictive impairment. An obstructive airway disease that can be described best as hyperactive airway disease occurs in individuals whose SCI occurs at T6 and above.

Heterotopic Bone Formation

Heterotopic ossification is the formation of new bone in soft-tissue planes surrounding a joint and it most commonly involves the hips. Presentation can include some combination of generalized or localized lower extremity swelling, loss of hip range of motion, fever, and elevated alkaline phosphatase level. Heterotopic calcification may occur subclinically and may be noted incidentally on radiographs.

Laboratory examination includes serum alkaline phosphatase, radiography, and bone scan. Bone scan may be positive before radiographic changes are noted. The main clinical problem (apart from distinguishing it from other problems, such as DVT, which can present similarly) is the loss of range of motion that can occur. Measures to possibly limit the eventual amount of bone mass formed include use of etidronate, nonsteroidal anti-inflammatory drugs (NSAIDs), and irradiation.

Aging

Spinal cord injury patients age faster than the able-bodied population. Menter and Hudson have developed a model of aging that predicts functional decline of the individual with duration of SCI as a result of the many changes that have been discussed. This model consists of the following 3 phases:

The acute restoration phase occurs immediately following SCI when the individual goes from having very little functional capabilities to regaining the maximum amount of functional return during rehabilitation. This phase is followed by ***the maintenance phase***, which is variable in length of time where the individual with SCI can enjoy a relatively stable level of function. This period is followed by a predictable ***functional decline*** that occurs as a result of degenerative effects of overuse syndromes and the physiologic aging process.

CHAPTER 22

Rehabilitation in Spinal Cord Injuries

Once a spinal cord and spinal column injury has been diagnosed and treated, rehabilitation begins. Rehabilitative efforts include physical, occupational, vocational, speech and recreational therapies and interventions will be more fruitful if family members and other caregivers are included. The duration of inpatient rehabilitation varies according to multiple factors, including neurologic level of injury, impairment below the level of injury, comorbidities, the patient age, premorbid functioning, motivation, social support and, most important, the functional goals for the patient. Inpatient treatment is only one step in a structured rehabilitation process that continues for months after hospital discharge. One should remember that rehabilitation is a lifelong process and becomes less structured after about the first year as patients adapt to their impairments, social situations change, and aging creates new functional challenges.

SKIN CARE

Skin care is an essential component of health maintenance in patients with spinal cord injury. The consequences of inadequate attention to this aspect of care can be devastating as recurrent skin breakdown results in limb amputation, osteomyelitis, sepsis, or death. The integumentary system is at risk in patients with spinal cord injury because of lack of protective sensation, decreased soft-tissue bulk, histologic skin changes, impaired mobility, and sometimes urinary or faecal incontinence. High-risk areas are bony prominences that receive high pressure during sitting or recumbency, such as the sacrum, ischial tuberosities, greater trochanters, heels, and occiput.

The key to prevention is patient education and the mainstay of prophylaxis is regular weight shifting, which may be performed by the patient with or without assistive equipment or by an attendant. In addition, the skin must be checked daily to allow early identification and treatment of pressure sores and to avoid progression to overt ulceration.

PRESSURE ULCERATION

Pressure ulceration can be the most limiting sequelae of SCI and can confine an otherwise independent individual to bedrest. Pressure to soft tissue above capillary pressure is the principal cause of skin breakdown. Shearing forces will lead to prolonged displacement of soft tissue relative to the underlying bone, which can distort interposed blood vessels and also can lead to tissue breakdown. Shear can occur over the sacral area when a person sits at an angle in bed with the bed surface fixing the skin at one point and gravity causing descent of the underlying sacrum. Evaluation includes an assessment of the ulcer depth. Destruction of tissue often is more extensive beneath the surface, and breakdown indeed may begin here rather than at the skin level. The location provides information about the cause. Ischial ulcers typically are due to sitting for prolonged periods without adequate pressure relief. Trochanteric ulcers are commonly due to excessive lying on side. Sacral ulcers, if high, may be due to supine lying. Lower ulcers in the intergluteal area may be related to sitting up at an angle. Prophylaxis involves limiting pressure and the time over which pressure is applied. Wheelchair and bed cushions that limit pressure in any one area by distributing it evenly over the available body surface are available.

Treatment of an established ulcer

Limiting or eliminating pressure to the area and removal of necrotic tissue is followed by cleansing with normal saline solution Topical antibiotics are used only for foul wounds. Proper nutrition, including adequate provision of calories, protein, vitamin C, and zinc. Smoking slows healing. Deep ulcers can be treated surgically with debridement and repair by myocutaneous flap.

NEUROPATHIC PAIN

Neuropathic (spinal) pain following SCI is perceived at or below the level of injury. Descriptors often involve temperature (e.g., hot, burning,

sunburned, frostbitten) and electricity (e.g., like an electric shock). Pain can exist apart from any external stimulus (rest pain) or can result from a stimulus that would, under normal conditions, not cause any pain (allodynia), or pain can be excessive in response to a painful stimulus (hyperalgesia). These symptoms may result from changes in central neuronal function, including increased spontaneous activity and reduced thresholds of response. If there is any change in an established pattern of neuropathic pain, suspect other unrelated disease process (e.g., renal stone). Medicinal treatment includes the use of anticonvulsants and antidepressants. Anticonvulsants may be particularly useful in cases of lancinating electrical pain. Gabapentin (initial dose of 100 mg PO tid, gradually titrated upward) typically is used, with precautions for sedation. Tricyclic antidepressants (amitriptyline [initial dose of 10 mg PO qhs, gradually titrated upward]) may be useful for more constant diffuse pain. Precautions must be taken for its anticholinergic effects. Patients should be informed that relief with these agents may not be immediate, as the initial dose may require modification, and in any case, the effect of the medication may not be apparent for days or weeks.

BLADDER MANAGEMENT

The goals of bladder management are to maintain continence and prevent complications while minimizing the negative impact on the patient lifestyle. While planning rehabilitation protocol, upper limb function, sexuality, social support, and personal preference must be considered and patient evaluation should include investigation of both the upper and lower urinary tract. SCI typically is followed by a period of bladder flaccidity. With suprasacral injury, reflexes eventually return. However, these reflexes may be unable to cause efficient voiding because of the tendency of reflex sphincter activity to directly oppose reflex detrusor contraction. This opposition occurs because of the isolation of the urinary tract apparatus from higher centers, which normally coordinate reflex activity. This problem is called detrusor-sphincter dyssynergy. Acute bladder management is by use of an indwelling catheter, as the bladder is likely to be flaccid. Intermittent catheterization is not practical during the initial phase when urine output cannot be controlled and is likely to be high due to the administration of intravenous fluids.

Long-term Management

Objectives

- A drainage method that is socially acceptable and that avoids wetting the skin
- Ensure complete bladder emptying
- Avoid high residual volumes

Selection of a bladder drainage method ideally is made following urodynamic evaluation and chronic use of an indwelling catheter is to be avoided when possible, as it can cause various complications (i.e., urinary tract infection, soft-tissue problems, renal problems). Clean intermittent catheterization is a method available to those with good hand function or to skilled attendants. The patient can be instructed to limit fluid intake, and catheterization is performed every 4–6 hours. Reflex bladder contractions, which could cause high storage pressure and incontinence between catheterizations, can be inhibited by agents such as oxybutynin (5 mg PO tid) or tolterodine (2 mg PO bid). Reflex voiding into a condom catheter is an option available to men with reflex bladder contractions. Voiding pressure sometimes can be decreased by alpha-blocking agents such as terazosin (initial dose 1 mg PO hs, titrated upward) or tamsulosin (0.4 mg PO qd).

NEUROGENIC BOWEL MANAGEMENT

Neurogenic colonic dysfunction is a particularly distressing and limiting impairment for a substantial proportion of those with SCI. Lower motor neuron dysfunction, as with cauda equina and conus medullaris injury, causes constipation with slow colonic transport and incontinence due to a flaccid sphincter mechanism. Upper motor neuron dysfunction also causes constipation with slow colonic transit and stool retention due to spasticity of the sphincter apparatus. However, with upper motor neuron injury, reflexes allowing defecation may remain functional. The goals of bowel training program are continence and convenience. Treatment regimen includes diet, specified fluid intake, oral medication, medication per rectum, timing, and positioning. The steps involved in establishment of a bowel program are evaluation, preparation of the patient, trials of a specific bowel program, and finally, adjustment of the program. Evaluation includes obtaining patient history to determine any pre-injury problems or patterns. Neurologic assessment, with examination

of the bulbocavernosus and anocutaneous reflexes, can suggest the presence of upper or lower motor neuron bowel dysfunction. Patients presenting with problems, including diarrhoea, often are impacted. This can be suggested by physical examination and can be confirmed by radiographs. Patients may be taking medications (e.g., antibiotics) that can have unintended effects upon the bowel, such as diarrhoea. Regulation of stool consistency is crucial and is best achieved through diet, but the aid of oral stool softeners, bulk laxatives, or both, may be needed.

Short colonic transit time

Intervention includes maintenance of adequate intake of fluid and fiber, with fiber acting as a sponge to hold moisture within the stool. Docusate sodium (100 mg PO bid) can increase the ease with which water enters the stool. Patients with lower motor neuron dysfunction may experience greater continence with stool that is firmer than would be optimal for patients with upper motor neuron dysfunction.

Prolonged colonic transit time

Intervention includes maintenance of adequate stool bulk, which stimulates contractions of the colon. Fiber is helpful with this. A bowel stimulant (e.g., 2 senna tablets PO qd) can be effective. These tablets typically are taken 8 hours before planned bowel evacuation. As these measures decrease bowel transit time, stool consistency may become softer.

Incontinence

The goal is to establish a set time for daily bowel evacuation, ideally after a meal to take advantage of any gastrocolic reflex that may be present. Specific evacuation strategies may differ for upper and lower motor neuron problems. With upper motor neuron injury, defecation can be triggered with application of an irritant to the anorectal area, such as stimulation with a gloved finger or application of a bisacodyl enema or suppository. With lower motor neuron bowel dysfunction, evacuation may be by use of the Valsálva maneuver and digital removal. In either case, emptying is facilitated by a seated position on a commode, as opposed to lying on side in bed.

AUTONOMIC FUNCTIONS

High thoracic and cervical SCI can cause loss of supraspinal control of sympathetic activity with dysregulation of functions normally imparted by sympathetic mechanisms. Baseline sympathetic activity is low, though there may be hyper-responsiveness of peripheral sympathetic receptors. Clinical problems are most common in those with injuries to level T6 and above, as such levels isolate the sympathetic outflow to the splanchnic vascular bed. Resting blood pressure is low with higher cord lesions, which is asymptomatic. Orthostatic blood pressure changes can cause weakness, lightheadedness, and fainting. Management of these patients includes gradual mobilization, liberal sodium intake, use of a compression hose, and an abdominal binder. Fludrocortisone acetate (0.1 mg PO qd) can expand intravascular volume and therefore is helpful. Other problem is bradycardia following spinal cord injury and usually resolves after several weeks. Tracheal suctioning can exacerbate bradycardia and can cause asystole, perhaps through a reflex increase in vagal output. Symptomatic bradycardia can be treated with intravenous atropine or by a transvenous, external, or implanted pacemaker.

SPASTICITY

Spasticity is a velocity-dependent increase in muscle tone and occurs commonly following SCI and other types of upper motor neuron injury. Spasticity causes resistance to passive motion of the limbs, exaggerated deep tendon reflexes, clonus, and involuntary cocontraction of muscle groups. Spasticity occurs following complete and incomplete cord injuries. SCI usually is immediately followed by a period of flaccidity, with spasticity developing over subsequent weeks. Spasticity has desirable and undesirable effects. It can be used to assist with mobility, especially by those with incomplete injuries. It can improve circulation and may be useful for decreasing the risk of DVT and osteoporosis. Spasticity also can interfere with positioning, mobility, and hygiene, and spasms can be painful. The treatment of spasticity includes the elimination of exacerbating factors and regular muscle stretching. Less invasive methods typically are employed before the more invasive methods. Prevention and treatment of noxious stimuli, such as pressure ulcers, a urinary tract infection, a urinary tract stone, or an ingrown toenail and regular muscle stretching will help to reduce spasticity. In difficult cases, drugs can be used (Table 22.1).

Other treatment options include botulinum toxin injection (useful for treatment of problems caused by specific muscle groups), intrathecal baclofen, peripheral procedures (neurolysis and contracture release) and central ablative procedures (rhizotomy and myelotomy).

Table 22.1 Functional status and level of injury

Drug	Daily dosage range	Common side effects
Baclofen	5–200 mg (in divided doses)	Hypotonia Sedation/confusion Withdrawal syndrome
Tizanidine	2–36 mg (in divided doses)	Fatigue Dry mouth Sedation Elevated LFTs
Clonidine	0.2–0.6 mg (in divided doses)	Orthostatic hypotension
Diazepam	5–40 mg (in divided doses)	Sedation Cognitive effects Tolerance dependence
Gabapentin	200–3200 mg (in divided doses)	Sedation/fatigue Ataxia Dizziness
Dantrolene	25–400 mg (in divided doses)	Weakness Elevated LFTs

GAIT

Some degree of ambulation may be possible for persons with thoracic level complete paraplegia and no lower extremity function with bilateral knee-ankle-foot orthoses (KAFOs) and a walker or crutches. For patients with paraplegia, performing a sit-to-stand transfer is laborious, the gait is in a swing-to rather than a reciprocal pattern, and the gait velocity is slow. Most patients with no lower extremity function are not trained in gait. With an injury level of L2, active hip flexion and reciprocal gait becomes possible. With an injury level of L3, ambulation with ankle-foot orthoses (AFOs), rather than KAFOs, is possible; however, the hips remain unstable due to the lack of active hip abduction and extension. Therefore, bilateral canes or crutches or a walker must be used.

UPPER EXTREMITY RECONSTRUCTIVE SURGERY

Tendon transfer surgery offers the opportunity to utilize an innervated but nonessential muscle to provide a lost function. A person with a spinal injury level of C5 may have good shoulder control and strong elbow flexion. Active elbow extension is lacking, making overhead activity impossible. Such a person may benefit from a transfer procedure to the triceps tendon. One of the muscles available for transfer is the posterior deltoid. A person with an injury level of C6 may lack effective lateral pinch and may benefit from transfer of a muscle (e.g., brachioradialis) to the tendon of the flexor pollicis longus. Other procedures are available to provide active finger flexion and extension.

FUNCTIONAL NEUROMUSCULAR STIMULATION (FNS)

Electrical stimulation of intact peripheral nerves can bring about contraction in muscles paralyzed by upper motor neuron injury for exercise and for function. Stimulation can be achieved by transcutaneous, percutaneous, or implanted electrodes. FNS can be used in the upper extremity to provide lateral pinch and palmar grasp to persons with, for example, C5 and C6 tetraplegia. A totally implantable system is available with control by the position of the contralateral shoulder.

SEXUAL DYSFUNCTION

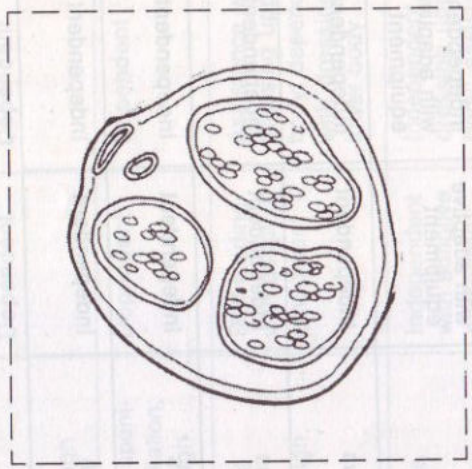
Sexual dysfunction must be systematically approached and treated with mechanical and pharmacologic interventions and psychosocial counseling. Procreation is also an important issue because semen quality and motility is reduced in men because of repeated urinary tract infections. Women may experience life-threatening autonomic hyperreflexia during delivery.

Table 23.1 Functional status and level of injury

Neurologic level	Respiratory function	Feeding	Dressing	Bowel & bladder function	Mobility
C1–C3	Mechanical ventilation; assisted cough	Dependent	Dependent	Dependent	Power wheel chair with respiratory control; dependent wheel chair transfers
C4	Potential for spontaneous ventilation; assisted cough	Dependent	Dependent	Dependent	Power wheel chair with chin control; dependent transfers
C5	Spontaneous ventilation; assisted cough	Self-feeding with adaptive equipment	Assistance with dressing upper body; dependent with dressing lower body	Dependent	Power wheel chair with hand control; dependent transfers
C6	Spontaneous ventilation	Independent with adaptive equipment	Independent with dressing upper body; assistance with dressing lower body	Potential for independence with orthoses or adaptive equipment	Manual wheelchair for short distances; potential for independent transfers with sliding board
C7	Spontaneous ventilation	Independent with adaptive equipment	Potential for independence with adaptive equipment	Independent	Manual wheelchair; independent transfers
C8-T1	Spontaneous ventilation	Independent	Independent	Independent	Manual wheelchair; independent transfers
T2-T10	Spontaneous ventilation	Independent	Independent	Independent	Manual wheelchair; ambulation with orthoses for exercise
T11-L2	Spontaneous ventilation	Independent	Independent	Independent	Indoor ambulation with orthoses; manual wheelchair for outdoor use
L3-S3	Spontaneous ventilation	Independent	Independent	Independent	Community ambulation with orthoses

contd.

Section 3 : Peripheral Nerve injuries



CHAPTER 23

Introduction

An important quality of the peripheral nervous system, as compared to the central nervous system, is its remarkable ability to recover after an injury through remyelination and regeneration of the axon. Technological advances in neurosurgical instrumentation and diagnosis have led to great results in the repair of acute nerve injury. Peripheral nerves are composed of afferent and efferent roots, anterior horn cells and interneurons, and the peripheral nerve itself. Damage to the peripheral nerves may affect motor and sensory fibers to different degrees. The neurological examination remains the cornerstone in evaluating a peripheral nerve condition, not easily supplanted by imaging advances as has occurred with most pathology affecting the central nervous system. A rigorous clinical approach allows the generation of an appropriate anatomical diagnosis. In addition to providing an understanding of the mechanism of injury or neuropathy, the history gives an appreciation of the temporal progression of the problem. This section will deal with the injuries of the peripheral nerves, approach to these patients and management of these injuries.

RELEVANT ANATOMY

Peripheral nerves comprise axons, associated Schwann cells enclosed in a basement membrane. Schwann cells ensheathe individual axons in myelinated fibers and groups of axons in unmyelinated fibers. The basement membrane is surrounded by thin collagen fibers called the endoneurium. The axon and Schwann cell composite is termed the endoneurial or Schwann tube. Endoneurial tubes are grouped together, forming a variable number of fascicles.

Perineurium surrounds each fascicle. Perineurium is composed of collagen fibers and concentric layers of closely packed flattened cells, united by tight junctions. The perineurium creates a diffusion barrier against the surrounding environment, similar to the blood-brain barrier. The perineurium maintains a positive intrafascicular pressure and protects against infection. Epineurium surrounds the layers of perineurium. The epineurium that fills the space between fascicles is called internal epineurium, and epineurium surrounding the nerve is termed external, or outer epineurium. The internal epineurium functions as a cushion for the fascicles. Where peripheral nerves span joints and where greater compressive forces are applied, the thickness of the internal epineurium is increased. The outer epineurial layers are composed of collagen and some elastin fibers. Anatomic fascicular groups are formed by condensations of internal epineurium. Interconnections between fascicles form a fascicular plexus.

CLASSIFICATION AND GRADING OF NERVE INJURY

Seddon's (1943) 3 grade (neuropraxia, axonotmesis and neurotmesis) classification scheme was based on clinicopathologic correlation, which was further expanded by Sunderland (1951) into 5 degrees of nerve injury. A comparison of the two systems is shown in Table 23.1 and characteristics of these injuries are described below.

Table 23.1 Classification of nerve injuries

Seddon	Sunderland
Neuropraxia	Grade I
Axonotmesis	Grade II
	Grade III
	Grade IV
Neurotmesis	Grade V

Sunderland Grade I (Neuropraxia)

- Reduction or complete block of conduction across a segment of a nerve with preservation of axonal continuity.
- No discernible histopathologic alteration in nerve except for localized thinning of axons, demyelination in severe cases.
- Motor more than sensory loss and sparing of autonomic function.
- Electrodiagnostic study results are normal above and below the level of injury.
- No muscle denervation changes are present.
- Tinel sign is absent.
- Recovery is complete.
- Recovery may take up to 12 weeks.

Sunderland Grade II (Axonotmesis)

- Axon continuity is disrupted but with relative sparing of the endoneurium.
- Wallerian degeneration distal to the level of injury and proximal axonal degeneration to at least the next node of Ranvier.
- Regeneration and reinnervation occur spontaneously.
- Electrodiagnostic studies demonstrate denervation changes in the affected muscles, and in cases of reinnervation, motor unit potentials (MUPs). Axonal regeneration occurs at the rate of 1 mm/day or 1 inch/month.
- Tinel sign present.
- Recovery occurs within weeks.

Sunderland Grade III

- Axon continuity is disrupted by loss of endoneurial tubes (the neurolemmal sheaths) but the perineurium is preserved.
- Wallerian degeneration occurs.
- Electrodiagnostic studies demonstrate denervation changes with fibrillations in the affected muscles. In cases of reinnervation, MUPs are present.
- Regeneration occurs at the rate of 1 mm/d.
- Tinel sign present.
- Regeneration and reinnervation may not occur.
- Recovery is mixed and incomplete.

Sunderland Grade IV

- Fasciculi are damaged but epineurium continuity is preserved.
- Large area of scar at the site of nerve injury and precludes any axons from advancing distal to the level of nerve injury.
- Electrodiagnostic studies reveal denervation changes in the affected muscles, and no MUPs are present.
- Tinel sign is noted at the level of the injury, but it does not advance beyond that level.
- No improvement in function is noted.

Sunderland Grade V (Neurotmesis)

- Endoneurium, perineurium, and epineurium, which make up the entire nerve trunk, are completely divided.
- Electrodiagnostic findings are the same as those for a fourth-degree injury.
- Recovery through axonal regeneration cannot occur.
- Requires surgery to restore neural continuity.

CHAPTER 24

Mechanisms & Pathophysiology

CAUSES OF NERVE INJURY

Blunt forces imparted to nerve remain by far the most common mechanisms of underlying nerve injury. There are several other mechanisms that can lead to nerve injury (Table 24.1).

Table 24.1 Causes of nerve injuries

- Entrapment
- Traction
- Stretch
- Contusion
- Laceration
- Compression a nd ischaemia
- Burns (thermal and electrical)
- Injection and iatrogenic injuries

Stretch Injuries

The internal anatomy of nerves permits the nerve to stretch approximately 10–20% before structural damage occurs. Failure of the axons, myelin sheath and connective tissue layers occurs in variable amounts, typically leaving the nerve in continuity with a variable spectrum of internal disruption. Rarely, a stretch insult may be so great as to result in complete mechanical failure of the nerve such that the nerve is literally pulled apart. In approximately half of these severe stretch plexus injuries, one or more nerve roots are avulsed from the spinal cord.

Stretch injuries or severe blows to a nerve cause axonotmesis. For example, forcible depression of the shoulder or traction of the arm can injure the brachial plexus. Other vulnerable nerves are described in Table 24.2.

Table 24.2 Causes of nerve injuries

Brachial plexus palsies	In infants, obstetrical complications In adults, following motorcycle accidents
Axillary nerve	Shoulder dislocation
Lumbosacral plexus	Severe fractures and dislocations in the pelvis
Sciatic nerve	Hip dislocation
Peroneal nerve	Knee dislocation

Laceration

Lacerations of the nerve can be caused by sharp or blunt objects and can lead to different degrees of nerve injuries (Table 24.3). In majority of cases missile or bullet does not actually divide the nerve. It produces intraneural damage secondary to shock, blast or cavitation effects. Late paralysis can also occur due to pseudoaneurysm formation and nerve compression.

Table 24.3 Causes of laceration injury

Sharp objects	Glass Scalpel Knife Razor blades
Blunt objects	Chainsaw injuries Metal and auto shrapnel Industrial accidents Propeller blade Low velocity missiles (i.e., as gunshot wounds)

Compression and Ischaemia

Concussion or compression of the nerve causes neuropraxia either by mechanical damage or by ischaemia. Relatively focal compression usually results in reversible nerve injury, whereas diffuse and longer duration compression, exceeding 8 hours, can lead to irreversible damage.

Causes

- Tourniquet paralysis
- Saturday night palsy resulting from mechanical compression as well as likely ischaemia of the radial nerve against the humerus.
- Anaesthesia related palsies
- Improper applications of plaster casts and splints
- Compression of nerves within a fascial compartment from haematoma

Compartment Syndrome

- Proximal venous ligation
- Direct trauma to muscle
- Infection
- Burns
- Localized pressure by casts or by circular dressings

Compartment syndrome injuries cause high pressure in the surrounding tissue. Pressure compresses the arterial blood supply of the nerve, predisposing the nerve to ischaemic cell damage and cell death. Even though the peripheral nervous system is relatively resistant to ischaemia, long periods of stretch and compressive force can cause vascular compromise and neuronal ischaemia. Delays in assessment and treatment of compartment syndrome can lead to nerve injury in the forearm.

Thermal and Electrical Injuries

Thermal and electrical injuries are relatively infrequent but cause severe and widespread damage. Several hours of non-freezing exposure with a temperature above–2.5 and below 10 causes damage to blood-nerve barrier and extensive endoneurial oedema leading to focal conduction block and cessation of axonal transport in the acute

stage, with early recovery of function. Severe cold injury like frostbite leads to necrosis of all involved tissues, including the peripheral nerves. Burns produce a direct thermal injury with neural damage ranging from a transient neuropraxia to severe neurotmesis Prognosis with most low voltage injuries is excellent, but quite variable for high voltage injuries.

Injection Injury

Serious complication of intramuscular drug administration. Sciatic nerve in the buttock is by far the most common one injected. Others are radial and axillary nerves. Damage may occur from the needle itself, but mostly is secondary to the toxic effects of the drug or agent being instilled in the intraneural compartment.

Needle placement results in an immediate electric-like shock sensation down the extremity; later, deep discomfort or bothersome paresthesias down the limb and in the distribution of the affected nerve; in 10% of cases, a delayed onset of neuropathy. Symptoms are often less dramatic. Neurological deficit may be complete or incomplete in the distribution of the injected nerve. When incomplete, motor loss is usually greater than sensory loss.

Other Iatrogenic Injuries

In many circumstances, it is not at all clear when the nerve injury actually arose. A relatively common situation is a fractured limb, where a delayed diagnosis of a neurological deficit is made. Following questions are to be answered while managing a patient with iatrogenic injury:

- Was the deficit originally present but missed because of the level of consciousness?
- Did the deficit occur as a result of action of the treating physician, such as closed reduction, application of a plaster cast or operative internal exploration and fixation?
- Only a good history and a reliable documentation with initial and serial physical examination would allow this to be answered, but is frequently unavailable. Nerves at relatively high risk are shown in Table 24.4.

Table 24.4 Nerves at relatively high risk for iatrogenic surgical damage

	Nerve	Causes
Upper extremity	Radial nerve palsies	Holstein-Lewis distal one third humeral shaft fracture
	Posterior interosseous nerve injury	Monteggia fracture-dislocation
	Ulnar nerve &/or anterior interosseous nerve injuries	Elbow dislocations
	Median nerve and/or radial nerve injuries	Supracondylar and medial epicondyle fracture in children
	Ulnar nerve injury	At the cubital tunnel
	Axillary nerve	Glenohumeral dislocation
		Proximal humerus fracture
		Direct blow to the deltoid muscle
Lower extremity	Peroneal division of the sciatic nerve	Traumatic hip dislocations
		Peroneal division is tethered at the fibular neck and at the sciatic notch and the tibial division is tethered only at the sciatic notch
	Common peroneal	Vascular surgery procedures such as arterial surgery
	Saphenous	Varicose vein stripping
	Femoral	

PATHOPHYSIOLOGY OF NERVE REGENERATION

Peripheral nerves respond to injury by a process called Wallerian degeneration.

Wallerian Degeneration

Wallerian degeneration occurs in peripheral nerves. It is a process by which the damaged segment of a nerve is phagocytosed, beginning at the first intact node of Ranvier. The Schwann cell tubes also are phagocytosed to prevent obstruction of the regenerating axon. Regeneration of a peripheral nerve occurs at the rate of approximately 1 mm/day. In injuries that are more proximal, improvement may not be obvious for many months. At the beginning stage of regeneration, the proximal axon stump sprouts buds that comprise the nerve growth cone. Axonal regeneration is guided towards the distal end of the nerve by a gradient of diffusible substances. This process is called neurotropism. Due to the constricting effect of intraneural and extraneural scar tissue, axonal regrowth can be blocked at the site of the lesion. NGF (nerve growth factor) affects the sensory regeneration but does not directly guide the regenerating axon. The axonal buds preferentially move toward neural tissue. However, they cannot differentiate between sensory and motor fascicles. The size of the distal fascicle appears to be the most significant factor in determining the target of the regenerating growth cone. Buds are more likely to find and attach to bigger fascicles and misdirected axonal buds can result in abnormal nerve connections. Abnormal motor nerve innervations can cause jerky or awkward movement. Motor endplates must be reinnervated within 18 months of trauma for function to be resumed.

CHAPTER 25

Evaluation of Nerve Injuries

EMERGENCY ROOM EVALUATION

In any trauma patient, attention to life-threatening airway, respiratory, circulatory and CNS injuries always takes first priority before limb injuries are addressed. In multi-trauma victims, nerve injuries and brachial plexus injuries are relatively frequent, affecting 5% and 1% of trauma patients, respectively. Moreover, these injuries can be diagnosed at the initial trauma encounter in over 60% of cases. In approaching the patient, a high index of suspicion is required. Any patient with a soft tissue, tendon, bony, joint or vascular injury in the limb should be examined for nerve damage. Conversely, patients with an obvious peripheral nerve injury should have careful assessment of their peripheral pulses and tendons, along with liberal use of radiological imaging of bones and joints adjacent to the area of nerve damage. The rule of thumb to exclude a nerve injury is to verify that the most distal aspect of the nerve is functioning. In patients with altered level of consciousness (even in the comatose patient), an asymmetric neurological exam, with loss of function confined to one limb when accompanied by loss of deep tendon reflexes can be suggestive of nerve injury. The precise distribution of nerve injury, even in the comatose patient, can also be ascertained by assessing for lack of autonomic function (sweating and loss of wrinkling after immersion of hand or foot in water). Purpose of initial neurological examination is:

- To exclude nerve damage
- To provide a baseline (as worsening function spontaneously and from iatrogenic circumstances [such as open or closed fracture reduction])

- Location and severity of injury
- Complete or incomplete nerve injury
- To look for preservation of any function (autonomic, sensory to autonomous distributions and especially motor).

Once the patients general condition is stabilized, further confirmation of nerve or plexus injury can be provided by detailed clinical examination and electrical studies, such as somatosensory evoked potential studies.

CLINICAL FEATURES

Pain

Pain is the frequent presenting complaint of patients with peripheral nerve damage. A common source of pain relates to disuse phenomenon (swelling, joint stiffness, muscle and tendon shortening and fibrosis) that follows many nerve injuries where the limbs are immobilized or not adequately mobilized. In cases of entrapment neuropathy, pain referred adjacent to and along the distribution of the compressed nerve is commonplace. For example, the description of aching discomfort in wrist and forearm, nocturnal symptoms and paraesthesias in the median nerve distribution are so characteristic as to be virtually diagnostic of carpal tunnel syndrome. Serious damage to peripheral nerves often produces an even more profound neuritic pain syndrome, characterized by severe, sometimes burning, pain in the distribution of the injured nerve, accompanied by sensory changes (hypoaesthesia and hyperaesthesia). Autonomic disturbance is characteristic of both true (or major) causalgia and minor causalgia (reflex sympathetic dystrophy). True causalgia typically occurs after significant damage (often from gunshot wound) to a major mixed nerve (such as median, ulnar and tibial). A severe burning pain, the patients careful attempts to protect the involved extremity from movement and manipulation, and evidence of autonomic over-activity are cardinal features. Some patients exhibit features of deafferentation pain, resulting from nerve root avulsion. Light touch or other non-painful stimuli may be perceived as painful (allodynia). Regenerating nerves may also produce pain, often described as tingling, electric shocks and dysaesthesias along the course of the nerve.

Sensory Loss

Sensory loss may be complete (anaesthesia) or a decrease (hypo-esthesia) in sensation. Enquire about functional consequences of sensory loss that may include accidental burning, injury and even frank ulcer formation.

Motor Deficit

Ask about any weakness of grip or any focal deficits. Questions directed as to how the patient performs on stairs or getting up from a sitting or squatting position will lead to improved understanding of the nature of the functional deficit. An obvious scenario is the baby with a plexus injury where information provided by the parents is particularly helpful. Much of this will simply reflect their day-to-day observations of the baby behaviour and play activity.

Autonomic Dysfunction

Autonomic disturbances may follow the major nerve injuries and it may be a hypofunction of hyperfunction (Box).

Autonomic hypofunction

- Loss of sweating
- Coolness
- Cyanosis
- Swelling

Sympathetic over-activity

- Sympathetically mediated pain syndromes
- Abnormal vasodilation
- Increased sweating

PHYSICAL EXAMINATION

After stabilizing the general condition of the patient, a detailed general examination will follow and before proceeding we should keep following questions in our mind:

- Is the lesion in the CNS (spinal cord, brainstem or brain) or can it be localized to a nerve root, or peripheral nerve distribution?
- What is the precise mechanism of nerve injury?
- What is the diagnosis?

- How severe is the injury?
- Are the mechanism and clinical findings consistent with a focal versus diffuse injury?
- Is the nerve element lacerated or in continuity?
- Is the damage to the nerve or nerve element complete or incomplete (sparing some aspect of autonomic, sensory or motor function).
- Grade of injury (neuropraxia, axonotmesis and neurotmesis).
- Is the patient condition static, improving or deteriorating?
- If the patient is worsening, a prompt search and correction of a secondary ischaemic or compressive (haematoma, compartment syndrome or pseudoaneurysm) complication is needed to prevent irreversible nerve damage.

General Principles of Peripheral Nerve Exam

General examination should be performed in a consistent and repeatable fashion so as not to overlook findings.

- A full exposure of the limb
- Compare one limb to the other
- Systematic and orderly approach, from proximal to distal limb (e.g., parascapular and shoulder girdle muscles before arm and forearm)
- Do examine joint movements (e.g., lateral abduction of the shoulder over the first 30 degrees is produced by supraspinatus, over the next 120 degrees by deltoid and then is completed by medial rotation of the scapula by parascapular muscles such as trapezius and rhomboids).
- Assess and grade individual muscles
- Be aware of and avoid being fooled by trick movements

Muscle Testing

Compare the involved and the unaffected extremity with respect to motor bulk, tone and then proceed to a thorough evaluation of strength of individual muscle groups. When possible, an accurate assessment of muscle bulk using a tape measure should be performed. In doing the latter, one should first mark the extremity from a fixed bony landmark so that one assesses comparable areas of the affected and the unaffected limb. When testing a given muscle, it is important to both inspect and be in a position to palpate the muscle being examined. This allows detection of even minimal flicker of contraction, which perhaps may not even result in limb movement.

Attitude

Observing the attitude of limb will provide valuable information about site of injury:

Erb palsy (upper and middle trunk brachial plexus injury)—The typical waiter tip posture is apparent, with the shoulder internally rotated and held tight against the body (as deltoid and spinatei are absent), elbow extended (as biceps is paralysed) and forearm pronated with the hand facing backward (as supinator is paralysed) with palm up (unopposed finger flexion from extensor paresis).

A radial nerve injury—produces a typical wrist and finger drop.

Posterior interosseous palsy—produces a characteristic finger drop only with wrist extension spared in a radial direction as extensor carpi radialis longus and brevis are spared.

Anterior interosseous nerve palsy—failure to make an 'O' when the tip of the thumb and index finger are brought in apposition as flexor pollicis longus and flexor digitorum profundus to index finger are affected.

Ulnar nerve injury at elbow—produces clawing of the fingers, which indicate loss of lumbrical muscle action, which normally flexes the fingers at the metacarpal phalangeal (MCP) joints and extends them at the interphalangeal (IP) joints. Loss of this function and unopposed long extensors (acting at the MCP joint) and the long flexors (acting at the IP joint) bring the finger into a claw configuration.

Distal median nerve injury—produces the Benedictine hand where the index and middle finger are similarly affected.

Combined forearm level ulnar and median nerve injuries—produces ape hand with all four fingers involved.

Sensory Evaluation

Sensory examination includes testing for light touch, pinprick, two-point discrimination, vibration and proprioception. In complete peripheral nerve injuries, all modalities of sensation in the distribution of the nerve will be lost; however, in incomplete or partial injuries some modalities may be affected more so than others.

To get a very quick idea about an area of anaesthesia or markedly altered localizing sensibility, the patient is directed to close his eyes and instructed to point to areas which are stimulated using light touch from the end of a blunt object such as a dull pen tip. This simple technique readily allows one to map the area of poor or absent sensation. One technique is to gently stroke the patients affected finger or area of sensory alterations simultaneously using the same force of finger stroke on an unaffected area and have the patient simply comment on the alteration of sensory touch.

Autonomous zones of innervation

A key principle regarding sensory testing for peripheral nerve injuries is to examine autonomous zones of innervation, where there is the least or no likelihood of sensory overlap from adjacent nerves.

Ulnar nerve—Volar aspect beyond the IP joint of the little finger.

Median nerve—Volar aspect beyond the distal IP joint of the index and the IP joint of the thumb.

Radial nerve—Anatomical snuffbox; however, there can be variable overlap from other cutaneous nerves such as the lateral antebrachial cutaneous nerve.

Reflexes

Always compare the affected to the unaffected side as myotatic reflexes are extremely sensitive indicators of peripheral nerve pathology. For example, it is not uncommon to find loss or diminution of the ankle reflex in a patient with a buttock or mid-thigh level sciatic nerve injury who has a complete peroneal division involvement clinically, but with nothing else to find (except an absent ankle reflex) with respect to the posterior tibial division. Further, once lost, myotatic reflexes often do not return even though peripheral sensation and function in the muscle might.

Autonomic Activity

Inspect the whole limb and digits for autonomic activity and look for:

- Colour
- Temperature

Table 25.1 Summary of physical examination

Nerve	Site of injury	Motor function	Sensory function
Radial nerve	Radial groove of humerus	Typical 'wrist drop' Extension of elbow, wrist, knuckles and all joints of thumb Supinator and brachioradialis	Sensory loss involves dorsum of 1st, 2nd and 3rd metacarpals May be as small as the anatomical snuffbox
Median nerve	Often injured by penetrating wounds of the forearm	The pronators Radial flexor of the wrist Flexors of all of the proximal interphalangeal joints Flexors of the terminal joint of the thumb, index and middle finger Abductor and opponens pollicis	Sensory loss over thumb, index, middle and half of ring fingers
Ulnar nerve	Fractures of the medial epicondyle of the elbow	'Claw hand' & hypothenar wasting due to loss of ulnar flexor of the wrist Flexors of the terminal phalanx of the ring & little finger Muscles of the hypothenar eminence Adductor pollicis Palmar brevis All the interossei and the medial two lumbricals	Sensory loss over little & half of ring finger

contd.

Erb's palsy	Due to damage to the upper nerve roots Usually involves C5, C6, C7	Abductors and external rotators of the shoulder are affected Loss of finger extension	Sensation is intact
Klumpke's palsy	Due to damage to the lower nerve roots Usually involves C8, T1	All finger muscles are paralysed	Loss of sensation often associated with unilateral Horner's syndrome

- Sweating behaviour (or lack of)
- Atrophic changes in skin organs and nail beds

Tinel Sign

An injured nerve often exhibits an overlying mechanical hypersensitivity. First discovered by Tinel, the finding of shock-like electrical sensations or paraesthesias evoked in the nerve distribution by percussing over the injured nerve is labeled as Tinel sign. A Tinel sign is often present overlying the nerve in an area of entrapment neuropathy and can remain present for long periods, sometimes indefinitely, overlying the area of previous nerve injury.

When a Tinel sign is found to be advancing along the anatomical distribution of the nerve, particularly if it does so at the expected rate of nerve regeneration, approximately 1 mm a day or an inch a month, then this provides evidence of ongoing regeneration. While an advancing Tinel sign may be a positive indicator of regeneration, it is associated with subsequent muscle reinnervation with functional recovery in only approximately half of the patients.

Hence, the patients need to be followed closely, even in the presence of an advancing Tinel sign. On the other hand, the lack of an advancing Tinel sign is a strong negative finding, suggesting complete neural interruption or poor regeneration. Some other tests are described such as Phalen test for carpal tunnel syndrome and modified Adson's test for thoracic outlet syndrome.

IMAGING STUDIES

Radiography

Many peripheral nerve injuries can be associated with other soft tissue or bone injuries that can be detected through radiographic findings (Table 25.2). Radiographs of the injury site help identify fractures or foreign bodies.

Table 25.2 Site of fracture and nerves involved

Cervical spine fractures	Brachial plexus
Unilateral elevation of the diaphragm	Phrenic nerve paralysis
Midhumeral fractures	Radial nerve injuries
Midforearm fractures of the ulna or radius	Median or ulnar nerve injuries, respectively
Hip and proximal femur fractures	Sciatic nerve injuries
Distal femur fractures	Peroneal or tibial nerve injuries
Supracondylar lucency (2 to 3 months after elbow trauma)	Delayed median nerve entrapment
Shoulder and cervical spine injury	Axillary nerve injury

MRI

On conventional MRI, signal changes in denervated muscles are seen as early as 4 days after injury and can be better seen with short tau inversion recovery (STIR) sequence. In nerve entrapment and neuropraxic nerve injuries, STIR or T2-weighted signals in the innervated muscles remain normal. Also, magnetic resonance neurography (MRN) can help visualize both normal and abnormal peripheral nerves in various regions of the body. It is useful in evaluating brachial plexus injuries.

Electrodiagnostic Studies

These objective tests are useful in detecting nerve injury and/or nerve compression and in identifying early stages of recovery.

Electromyography (EMG)

This test is performed at least 4 weeks following nerve injury. EMG testing done prior to that time may yield false-negative findings because it takes 4–6 weeks for muscle fibrillations to become apparent. Evidence of denervation is indicated by the presence of fibrillations in the muscle. Reinnervation is noted by the presence of motor unit potentials.

Nerve Conduction Studies

These studies are particularly useful in determining secondary compression sites that may be present. If the nerve is compressed at an entrapment site, such as the carpal tunnel or cubital tunnel, axonal regeneration may be impeded and thus limit reinnervation.

CHAPTER 26

Management of Nerve Injuries

Principles of tetanus prophylaxis, antibiotic administration, timely closure of lacerations and debridement of contaminated and extensive wounds are adhered to as in any other emergency situation.

GOALS OF MANAGEMENT

The goals of treatment of these patients are:

- To return function to the damaged nerve
- To improve the quality of life of patients
- Not only is the nerve treated, but exogenous sources of nerve injury also are treated
- Bone dislocation with neurological deficit requires prompt anatomical reduction to prevent irreversible nerve necrosis
- Analgesics to control pain
- Steroids to decrease endoneurial Oedema
- Protection of the joints, including the surrounding ligaments and tendons, from further stress

Splints, slings, or both—For example, a radial nerve injury results in a loss of wrist and finger extension, a wrist drop. A wrist-resting splint may be used to support the hand in a neutral wrist position and place the hand in a more functional position. In patients with brachial plexus nerve injuries, particularly when C5–C6 is affected, continued downward stress at the glenohumeral joint may cause the glenohumeral joint to subluxate without the muscle support of the rotator cuff muscles. A sling is helpful to unload this joint, prevent complete shoulder dislocation, and decrease pain.

INDICATIONS FOR SURGERY (TABLE 26.1)

Surgical intervention for acute nerve injury is based on the extent of damage to the nerve and the nerve functional viability. Consider each patient on an individual basis. When evaluating patients for surgery, surgeons should consider the location, the extent of the injury, the patient age, and the patient medical condition. Two important questions to consider before surgery are whether function can be obtained from the repaired nerve and whether the potential benefit to the patient outweighs the surgical risks, costs, and loss of productivity. For example, adults older than 40 years rarely achieve a functional result from ulnar nerve repairs proximal to the elbow. Consequently, these patients may not be candidates for surgery. When certain cases are unresponsive to conservative treatment, surgery is the only alternative. If nerve function is progressively deteriorating as per electrodiagnostic study findings, surgery may be indicated because the status of the connective tissue cannot be assessed without direct exploration.

Table 26.1 Indications for surgery

Closed injuries	No evidence of recovery either clinically or with electrodiagnostic studies at 3 months following injury
Open injury (i.e., laceration)	Surgical exploration is recommended as soon as possible All lacerations with a reported loss of sensation or motor weakness
Crush injury	Repair may be delayed for as long as several weeks If after 3 months no evidence of reinnervation (electrically or clinically)-surgical reconstruction with repair or graft is indicated
Grade 5	Early surgical repair
Location of nerve injury	The more proximal nerve injuries and those distal injuries not exhibiting spontaneous recovery need exploration

SURGICAL THERAPY AND TIMING

Sharply Divided Nerves

In situations of sharp penetrating trauma with nerve injury, i.e., sharply divided nerves, a primary exploration and suture repair of the divided nerve, aided by microtechniques and magnification, in the operating room is best. This repair is to be performed within hours to a day or two.

Lacerated Nerves

If the nerve is merely contused or bruised, it is best to make a careful note of the location and degree of damage, and then follow the patient clinically for evidence of recovery. Treatment of these injuries is delayed by 3–4 weeks following injury as this delay will allow the longitudinal extent of injury to be fully delineated so that debridement of the nerve can be done to healthy proximal and distal stumps before repair. Injuries that do not demonstrate evidence of early spontaneous recovery, such as those caused by bullets, crushing blows, traction, fractures, or injections, are explored 2 months after the injury. For a nerve injury within 2–3 inches of recoverable muscle, 2 months is required for the growing axons to begin the process of muscle reinnervation. Therefore, an additional delay of 1 month is justified before surgical exploration.

Brachial plexus stretches or contusions are observed for 4 months. If no evidence of recovery is present, the plexus is explored. Because nerves in the elbow are anatomically vulnerable, many researchers recommend early surgical exploration after fractures of the humerus. Justification for late exploration is to allow sufficient time for spontaneous recovery. For example, for patients without functional recovery of ***the radial nerve***, the time frame for late exploration ranges from 8 weeks to 5 months. This allows sufficient time for spontaneous recovery without jeopardizing the results of late repair. Injuries consistent with nerve rupture (e.g., glenohumeral dislocation), a 3- to 6-month period without clinical or electrophysiological evidence of recovery warrants surgery. This timeframe is sufficient for neuropraxic and axonotmetic injuries to resolve but is not long enough to jeopardize results of subsequent ***axillary nerve*** surgical repair. In the case of traumatic hip dislocation with a successful closed reduction, a short period of conservative treatment is recommended before surgical exploration.

SURGICAL TECHNIQUE

Best surgical results occur when the nerves are either purely sensory or purely motor and when the intraneural connective tissue component is small and the fascicles have been clearly aligned.

Primary Repairs

Sharp lacerations without loss of nerve substance or partial lacerations with proper alignment are good examples of injuries that benefit from primary epineurial repair with end-to-end anastomosis.

Secondary or Delayed Repairs

In a crushing or delayed repair requiring trimming of the nerve ends, group fascicular repair improves fascicular alignment without an excessive number of sutures. Secondary repairs are delayed repairs that may entail different strategies. Bones can be shortened to add length to a nerve. Nerve transposition across a flexed joint (e.g., the ulnar nerve in the elbow) is another strategy for gauging nerve length in secondary repairs. In addition, during a delayed repair, scarred ends of the nerve can be defined more accurately and trimmed back to normal fasciculi. The epineurial suture is more secure because the sheath has toughened. The suture of a severed nerve should not be delayed beyond 1 month. Neurolysis is performed on intraneural and extraneural scar tissue to release regenerating nerve fibers in the hope of improving functional recovery. Contaminated wounds, such as gunshot wounds and avulsions with severe tissue disruption, benefit from a secondary repair. Severely damaged nerves may require a nerve graft. The sural nerve is the criterion for nerve autografts because of a favourable ratio of axons to epineuriums. Loss of the sural nerve produces only a well-tolerated sensory loss on the lateral foot. For shorter nerve gaps, the anterior branch of the medial antebrachial cutaneous (MABC) nerve is a good nerve graft donor because the donor site scar is minimal and the resultant sensory loss is on the anterior aspect of the forearm. Extensive research has focused on the use of allograft nerves to replace peripheral nerves that require a long nerve graft. Allografts can survive if the patient is immunosuppressed and if the nerve allograft is preserved to maintain cell viability. Immunosuppression can be discontinued when the nerve graft has been incorporated with an in-growth of Schwann cells from the host

nerve ends. Nevertheless, results from autograft use are slightly more favourable than allograft use. Nerve or tendon transfers may be necessary for unrestorable or unsuccessful nerve repair. Brachial plexus injuries are not always repairable. In such cases, neurotizations or nerve transfers may offer a better functional outcome. The spinal accessory or long thoracic nerve can be grafted onto distal arm nerve trunks, with some improvement in elbow flexion. When repair cannot, or does not provide adequate results, planned tendon transfers can increase extremity function.

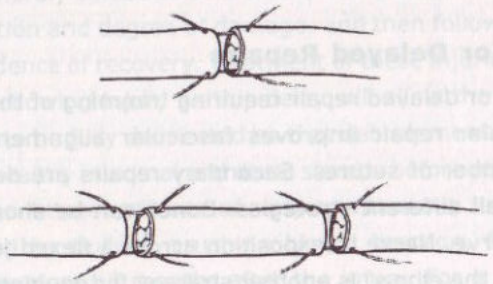


Fig. 26.1 Technique of nerve repair.
Direct repair (*upper*), with graft (*below*)

Tendon Transfers

Do not perform tendon transfers prior to 3 months after injury because early surgical exploration with nerve graft placement yields better results compared with primary tendon transfer. Tendon transfers, such as the posterior tibialis tendon passing through the interosseous membrane, can add power to a foot with a peroneal deficiency.

SPECIFIC CONSIDERATIONS

Lacerations

Sharp clean lacerations can be sutured primarily and a direct repair is performed. For bluntly lacerated nerves with more crushing or avulsion injuries, wait for clear demarcation to occur and the definitive reconstruction is done at 3 weeks or when the wound permits. Treatment of these lesions includes the evaluation in the operating room with appropriate magnification, and internal neurolysis, to spare the nerve element that is in continuity and selective repair of the divided nerve elements.

Traction, Stretch and Contusion

Timing of surgery continues to be somewhat controversial. It is generally accepted that most of these in continuity lesions should be explored between three to six months from onset of injury, in circumstances when they fail to spontaneously improve.

Compression and Ischaemia

A closed compartment syndrome presents another type of emergency situation, where prompt attention to decompression with thorough fasciotomies are needed to prevent an irreversible ischaemic infarction of muscle, nerve and other soft tissues, leading to a devastating Volkmann ischaemic contracture.

Thermal and Electrical Injuries

Initial management is directed towards treatment of the life-threatening circumstances and proper attention to wounds, with the nerve damage taking a much lower priority. Delayed deficits from compression by circumferential eschars can be avoided by prompt and thorough escharotomy. Generally, nerve grafting and repair carry poor results given the often extensive length of the damaged nerve and the poor vascularity of the soft tissue bed.

Gunshot Wounds

Typically, gunshot wounds associated with neurologic deficit have good potential for neurological recovery and 70% spontaneous recovery of function will occur over several months. Thus, unless an associated vascular or bony problem is present, the patient with a neurologic deficit following a gunshot wound is managed conservatively and monitored with frequent clinical examinations. Careful clinical and electrophysiological follow-up to offer exploration and possible nerve repair for patients not exhibiting evidence of recovery over approximately four months is worranted. With this approach, approximately 60% of patients with civilian gunshot wounds to the brachial plexus require nerve exploration, and many of them require nerve repair. Remember a baseline clinical examination is particularly important in the patient sustaining missile wounds, as there is a somewhat higher incidence of concomitant vascular injury with pseudoaneurysm formation. Such patients exhibit progressive

neurological loss and require urgent angiography to establish the diagnosis, followed by prompt repair of the aneurysm and/or expanding haematoma to prevent irreversible neurological deficit.

POSTOPERATIVE CARE

Part is immobilized in a bulky dressing for several days following surgery. The area of nerve coaptation then is immobilized for a longer time postoperatively (nerve graft for 10–14 d, nerve repair for 3 wk), although the patient is instructed in range of motion exercises for the joints proximal and distal to the immobilized region. For example, a median nerve repair at the wrist would be immobilized with a wrist-resting splint, and the patient would continue with range of motion for the fingers, elbow, and shoulder.

COMPLICATIONS OF ACUTE NERVE INJURY

A variety of pain syndromes can result. Plexus or root avulsions may produce burning dysaesthesias and paraesthesias. Painful neuromas and entrapment syndromes can occur. Partial nerve injuries of mixed motor and sensory function can lead to causalgia—symptoms include severe hyperaesthesia, hypersensitivity to cold or muscle activity, and increased pain in stressful situations. Paralysis can complicate nerve injury and sometimes cannot be repaired. If physical therapy is not instituted promptly after surgery, denervation can develop and result in muscle atrophy and fibrosis, joint stiffness, motor endplate atrophy, and trophic skin changes.

COMPLICATIONS OF NERVE SURGERY

In general, contraindications to surgery usually result when the risks of surgery outweigh the benefits. Surgery should not be performed when a poor outcome is expected. Complications of nerve surgery may be as in any other surgical procedures, including:

- Infection
- Haematoma
- Seroma
- Injury to surrounding structures, including vascular structures
- Further injuring the nerve, particularly in mixed nerve injuries.

PHYSIOTHERAPY

Physical therapy is started in the early stages following nerve injury to maintain passive range of motion in the affected joints and to maintain muscle strength in the unaffected muscles. Regular physical therapy helps to maintain a range of movement and to optimize the recovery of motor function as muscle reinnervation occurs. Before starting physiotherapy, protect repairs by relaxed joint posturing for approximately 3 weeks. Also, to prevent disruption of sutures at the repair site, the patient should avoid overzealous physical activity. In nerve transfers, the extremity is immobilized for 4 weeks after surgery, at which time physical therapy is initiated. In the later stages, sensory and motor reeducation is recommended to maximize the outcome.

PROGNOSTIC FACTORS

Prognosis depends on multiple factors, including:

- Trunk level or higher
- Patient compliance and motivation
- The extent of injury to neural tissue
- Contamination of the wound
- Age and medical status
- Surgical delay in excess of 5 months dramatically decrease the rate of functional return
- Neuropraxic injuries usually are reversible
- Associated injuries
- Amount of healthy proximal axon remaining after injury

FOLLOW-UP CARE

Clinical outcome is documented by serial clinical examinations and electrodiagnostic studies. Initially, the patient is monitored for postoperative wound healing. After immobilization and once the patient regains full passive range of motion, the patient is monitored every few months to evaluate for evidence of reinnervation. With nerve regeneration, a Tinel sign progresses distally along the nerve. With muscle reinnervation, a muscle contraction is visible, and with sensory reinnervation, the patient responds to light touch. Depending on the level of injury, the patient may continue to progress for varying periods—distal injuries more quickly and proximal brachial plexus injuries for 2–3 years following surgery.

As a general rule, it is suggested to examine patients at 2 weeks, 6 weeks, 3 months, 6 months, 1 year, and then at yearly intervals if necessary and practical after surgery.

Test and document range of movement and recovery of strength and sensation at each visit. Postoperative clinical and electrodiagnostic examinations are performed every 3 months for the first 2 years after surgery and every 6 months thereafter. Electrodiagnostic studies can help detect early signs of muscle reinnervation, several months before clinically evident muscle contractions appear. After nerve transfer surgery, assess patients 3 years after surgery. In most cases, maximal recovery requires as long as 24 months.

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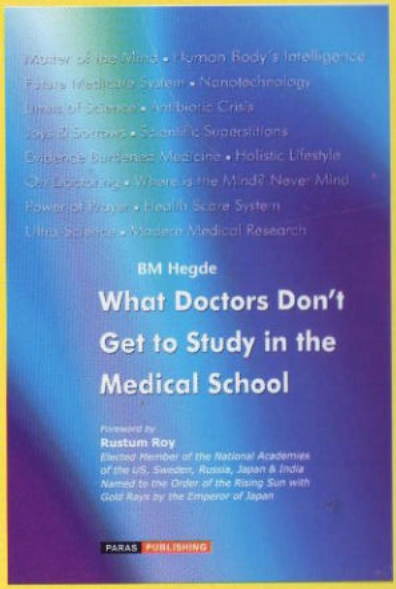

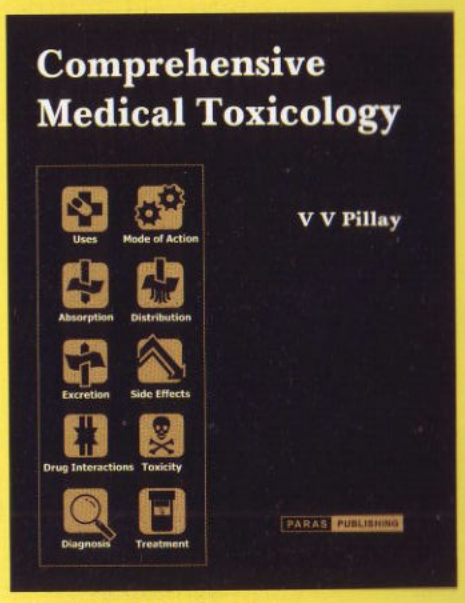

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